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- Anorexia Nervosa
- Antidepressants
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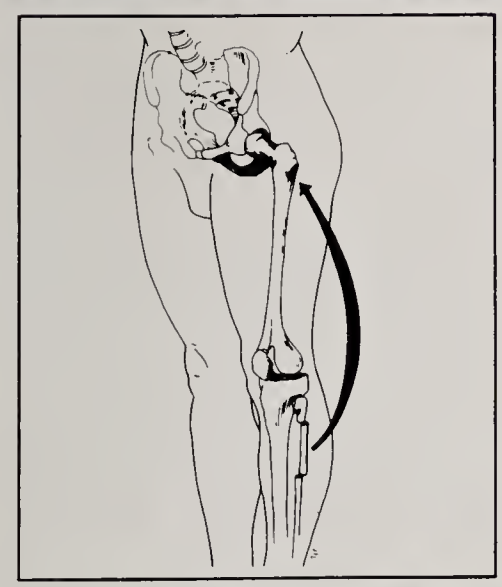
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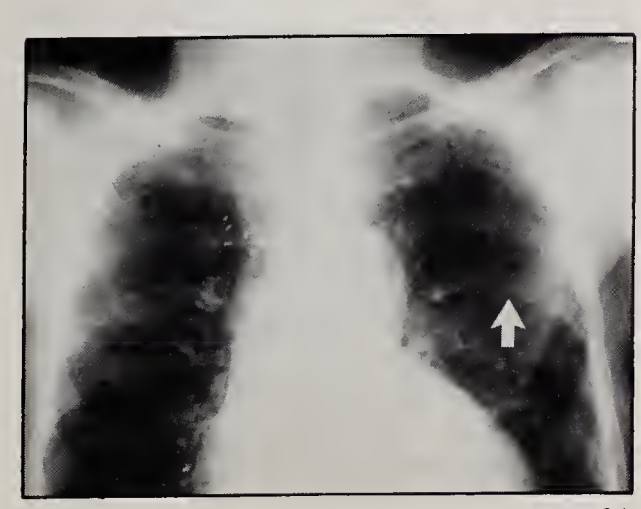
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Cover illustration:
Compulsive handwashing is a hallmark of Obsessive-Compulsive Disorder, illustrated here by Karen Visser, a second year Art as Applied to Medicine student.

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Editorial

Catherine Birt, Michael Paré and Raymond Ng
Editors-in-Chief

A few months ago, in the summer, members of the editorial board were working around the clock preparing the first issue. At that time, we were confronted with the difficult task of choosing the best articles from among the collection of well written, interesting papers submitted to us. When time came for the production of the second issue, we found ourselves once again facing an even more difficult situation. Not only had we received more good articles in the last few months (although not enough!), but we are also committed to improving the quality of each issue we publish.

The selection process has never been easy. The Journal is read by a broad spectrum of individuals with divergent backgrounds. However, the undergraduate medical student body continues to represent the largest share of our readership. With this fact in mind, we have elected to place some emphasis in this issue on undergraduate learning. We have, therefore, chosen a few articles on some common and interesting subjects that we believe will be useful to our fellow students.

One example of this is the article on Anorexia Nervosa, in which we learn about the various aspects of this fascinating psychological ailment. By using a case study, the author illustrates in great detail how this disorder can manifest itself clinically. We have also published the paper on the Elderly Gravida, which describes the different kinds of risks associated with pregnancy in older women. This is an important and significant issue, since women today not uncommonly delay the timing of their first pregnancy. Finally, we have also included our regular feature of Clinical Rounds, in this issue on an aspect in the field of Cardiology.

We hope that this issue will be interesting and beneficial to our readers, especially our fellow students.

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Anorexia Nervosa

Lawrie Graham, 8T9

Abstract

Anorexia nervosa is an eating disorder that has a potentially lethal outcome. It may begin as a seemingly harmless diet, but can quickly mushroom into an overwhelming obsession to lose more and more weight that controls the patient's whole life. In the following discussion, a case study is presented which illustrates many of the characteristics of the syndrome of anorexia. The etiology and pathogenesis, the impact on other family members, the treatment of the disorder, and the implications for society are also discussed.

Introduction

Anorexia nervosa is an eating disorder that is widespread in our society and growing at an increasingly alarming rate. Characteristics of the syndrome include excessive weight loss, an altered perception of the body image such that the sufferers believe their body proportions to be larger than they truly are, and a phobia or obsessive fear about becoming obese.[1] The condition usually affects women in the 12 to mid - 30's age range, although recent documentation suggests that the age group is widening to include children as young as 10 and adults in their 50's. More men than ever before are also falling victim to anorexia, the ratio now being 10 women to every 1 man.[2]

The onset of anorexia is often an insidious one. It generally begins in a teenager who perceives herself to be overweight in comparison to societies' ideal. As a result the young girl begins a seemingly innocent diet which progresses into an uncontrollable obsession to be thinner and thinner. In order to lose weight, all anorectics severely restrict their caloric intake. It has been estimated that the typical average daily intake of anorectics is between 200 to 900 calories.[3] Along with self-starvation, many anorectics exercise to the point of physical exhaustion in order to lose extra weight. In addition, self-induced vomiting, and the use of laxatives and diuretics is also quite common.[1]

Etiology

Many theories have been proposed to explain the pathogenesis of anorexia nervosa, including biological, psychological and sociocultural models. No single model appears to be sufficient; rather a variety of causative factors may work together in the development of the syndrome. For example, western society places a very high emphasis on being thin and attractive. In a 1971 survey of 1241 school girls aged 14 and older, it was discovered that half of the girls believed themselves to be overweight, and one third had attempted dieting to reduce their weight.[3] This cultural overemphasis on physical appearance may be one causative factor in the development of anorexia.

Psychological predisposing factors in the development of anorexia include the fact that many sufferers have distorted concepts and attitudes: one must strive to be perfect, only thin people are good and worthy of love, gaining weight means one is bad and out of control. These perfectionist tendencies and the desperate need to be in complete control of all aspects of their lives become manifest in the stringent control of their eating behaviour. Also within this realm of psychological dysfunction is the observation that there is a very high incidence of depressive symptoms in patients with anorexia. One study estimates that up to 50% of people with anorexia meet the criteria for a major depressive disorder.[1]

The final piece of insight into the etiology of anorexia comes from family studies which suggest that many of the patients come from socially maladjusted families in which there is a high degree of discord between the parents.[3] To the casual observer these families usually appear to be harmonious, but once the surface has been cracked the dysfunctional relationships that are uncovered may be an important contributing factor to the development and maintenance of anorexia.[1]

Treatment

Parents of children with anorexia feel very frustrated when their repeated attempts to force their child to resume normal eating behaviour fail. Parents are used to being in control of their children's lives — what time they go to bed, what clothes they wear, what they eat — and this loss of control puts them in a very disadvantaged position. They feel helpless and inadequate as parents as they watch their child deteriorate further. Parents are placed at a further disadvantage once the child is admitted to hospital. In this setting the parents are forced to relinquish control of the child to the attending physician. Often the parents know very little or nothing about the syndrome of anorexia nervosa and must rely on the doctor to make responsible decisions concerning the welfare of their child. To alleviate much of the anxiety the situation causes the parents, it is the responsibility of physicians to keep the parents well informed about their child's progress and to include them in as much of the decision making process as possible.

Since psychological, sociocultural and biological factors are all considered to be of importance in the etiology of anorexia nervosa, treatment of the disorder can be extremely complicated. The role of the physician is to organize a treatment regime utilizing the many different specialties available, including psychiatrists, nutritionists, social workers and other health professionals. Generally the patient must be admitted to hospital in order to ensure adequate weight gain and nutrition. At that time, in addition to medical therapy, psychological therapy is routinely begun which includes group

therapy, family therapy and individual therapy.[3] Even after the patient has been discharged from the hospital and therapy is complete, the physician's role is not complete. It is imperative that the physician perform routine follow-up evaluations since it is well known that over one third of patients have recurrent episodes of anorexia.[1]

Implications for Society

The ever expanding population of people with eating disorders has grave implications for society, especially with regard to the funding and organization of health services and treatment facilities. The latest American statistics show that one in every ten people has bulemia and one in every 100 has anorexia. The Canadian statistics are probably very similar. The British Columbia

Medical Association financed a study which showed that there were six times as many people with eating disorders in 1986 as there were in 1983.[2] With the incidence of anorexia increasing at such an alarming rate, there are simply not enough doctors who are adequately trained in the treatment of anorexia. There are also not enough hospital beds or treatment facilities to deal with the increased case load.

Dr. John Fleming, co-ordinator of The Hospital For Sick Children's eating disorder service states: "We're treating twice as many patients as we can comfortably handle. We're approaching the point where we'll have to close our doors for a while and accept no new patients".[2] The thought of having to refuse treatment to a person in need is disconcerting, especially given the fact that 2-5% of patients with chronic anorexia nervosa attempt suicide.[1] In Ontario there are no government funded clinics for the treatment of adolescent eating disorders and the adult treatment centres are operating at maximum capacity. Specialists operating private facilities charge from $65 per hour and up.[2] The majority of people cannot afford to pay this price for the large amount of treatment necessary to effect a cure. It is obvious that the growing number of people with eating disorders will have important implications for society concerning the organization and funding of health services.

Case Study

In order to obtain a more personal perspective into the social and psychological impact of anorexia nervosa on both the patient and the family members, the case history of a 22 year old woman, Karen G., is presented. At 12 years of age, Karen was a slightly overweight young girl. For years her family and friends had made jokes about her weight calling her 'fat moo-moo G.' and 'little butter ball'. In an attempt to conform to their expectations of what her ideal weight should be, she bought a diet book for teenagers. She only ate half of the suggested diet thinking this would allow her to lose weight faster. Friends and family congratulated her when she began to lose weight. At school, friends would ask her for a copy of her diet. The compliments and the extra attention paid to her made her believe that people liked her better when she was thin, thus encouraging her to lose even more weight. As the weeks passed by she ate less and less until a point was reached when all she consumed in a day was a glass of milk and two pieces of dry toast. At meal time with the family she would play with her food spreading it out on the plate to make it appear as if she had been eating, or she threw it into the garbage when no one was looking. Over a six month period, Karen went from 101 lbs. to 65 lbs. She was 5 feet tall. On admission to the hospital she was eating no food and drinking no water for fear of gaining weight.

As well as restricting her food intake, Karen also exercised ritually. Every time she finished eating a meal she would run up and down the stairs in her house until the point of physical exhaustion. When walking outside, she made it a rule that her feet could only touch pavement. Thus when her friends took a short-cut across a field or a neighbour's lawn, Karen always took the longest route around on the pavement by herself because the extra walking burned up more calories. During the breaks between periods at school, Karen walked around the perimeter of the classroom instead of socializing in an attempt to burn off all the calories that she could in the time available. The ritual of exercise caused her to become socially isolated from her friends.

Severe personality changes also contributed to Karen's social isolation from her friends and family. Previously a boisterous and fun loving girl, she became quiet and despondent. She recalls feeling very little emotion at the time — "even smiling was difficult". She began to concentrate on being the perfect little girl — perfect handwriting, perfect grades at school, perfect weight — in a desperate attempt to please others. Karen reported that "I became a 'yes-girl' because if it made someone happy I wanted to do it", and "I just wanted to please people". She refused to go out and play with her friends preferring instead to stay home and do the housework both to please her mother and because it was exercise.

Watching Karen slowly starving herself to death had a traumatic effect on the rest of the family. Her parents began to get very concerned as they watched Karen eating less and less as the weeks passed by. They went to the family doctor for some advice on how to best deal

with the situation. He suggested that they not force Karen to eat for fear that this would cause her to retaliate by restricting her food intake to an even greater degree. Initially her parents complied but as the weeks passed and Karen continued to lose weight, Mr. and Mrs. G became frantic with worry and started forcing Karen to eat. Her mother would back her into a corner of the kitchen and physically force food into her mouth. Her father too would sit with her at meal times and not allow her to leave the table until she had eaten some of the food on her plate. After these episodes Karen would exercise frantically in an attempt to burn off all the excess calories her parents had forced her to eat. After a lot of sleepless nights spent worrying and a lot of tears, Karen's parents finally had her admitted to The Hospital for Sick Children.

During Karen's treatment at the hospital, the doctors attempted to ascertain the causative factors which led to the development of anorexia. Karen had demonstrated many distorted concepts and attitudes in the three years preceding the development of anorexia and these continued to the end of her treatment. Initially she went through a phase where she apologised for every little mistake she made, to the extent that she would say "sorry" to a table if she accidentally kicked it. As this phase ended she began a new phase where she felt compelled to wash her hands repeatedly throughout the day because she believed that everything she touched was dirty. After this phase, she became obsessed with the fact that her face was not symmetrical. She spent hours in front of the mirror examining her facial structure and doing facial exercises in an attempt to make her face perfectly symmetrical. These perfectionist tendencies may have been related to the development of anorexia.

Karen's homelife was also important in the etiology of the syndrome. She recalls that her parents were continually fighting before she had anorexia to the extent that she was embarrassed to bring friends home. Once her parents realized the seriousness of Karen's problem, they stopped fighting and focused all their attention on her. Cessation of the arguments acted as a reinforcement and encouraged Karen to maintain her abnormal eating behaviour.

Conclusion

Anorexia is a disorder of great public health concern both because of its frequency and its extremely disabling consequences, particularly in relation to the psychological and emotional functioning of its sufferers. It is a disorder which has been attributed to many causes, all of which seem to have some validity. It is likely that many factors combine to contribute to both the establishment and maintenance of the syndrome. Far more extensive research into the pathophysiology and treatment will be necessary in the coming years.

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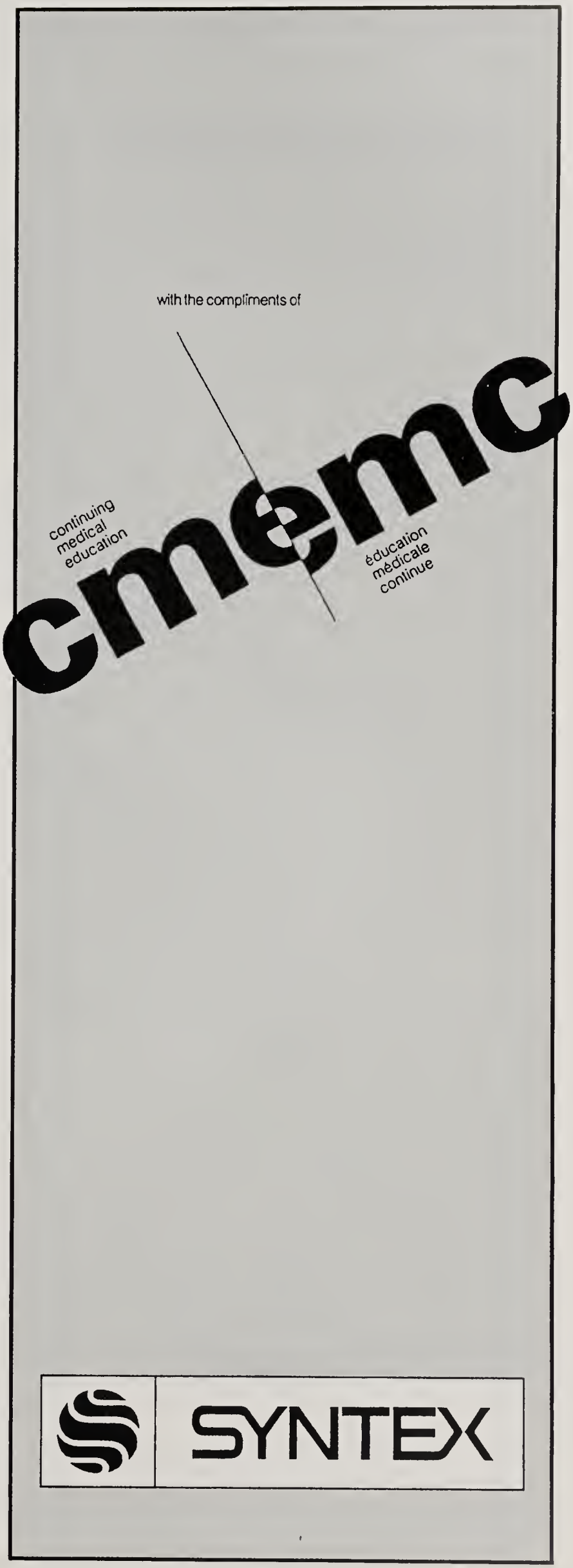
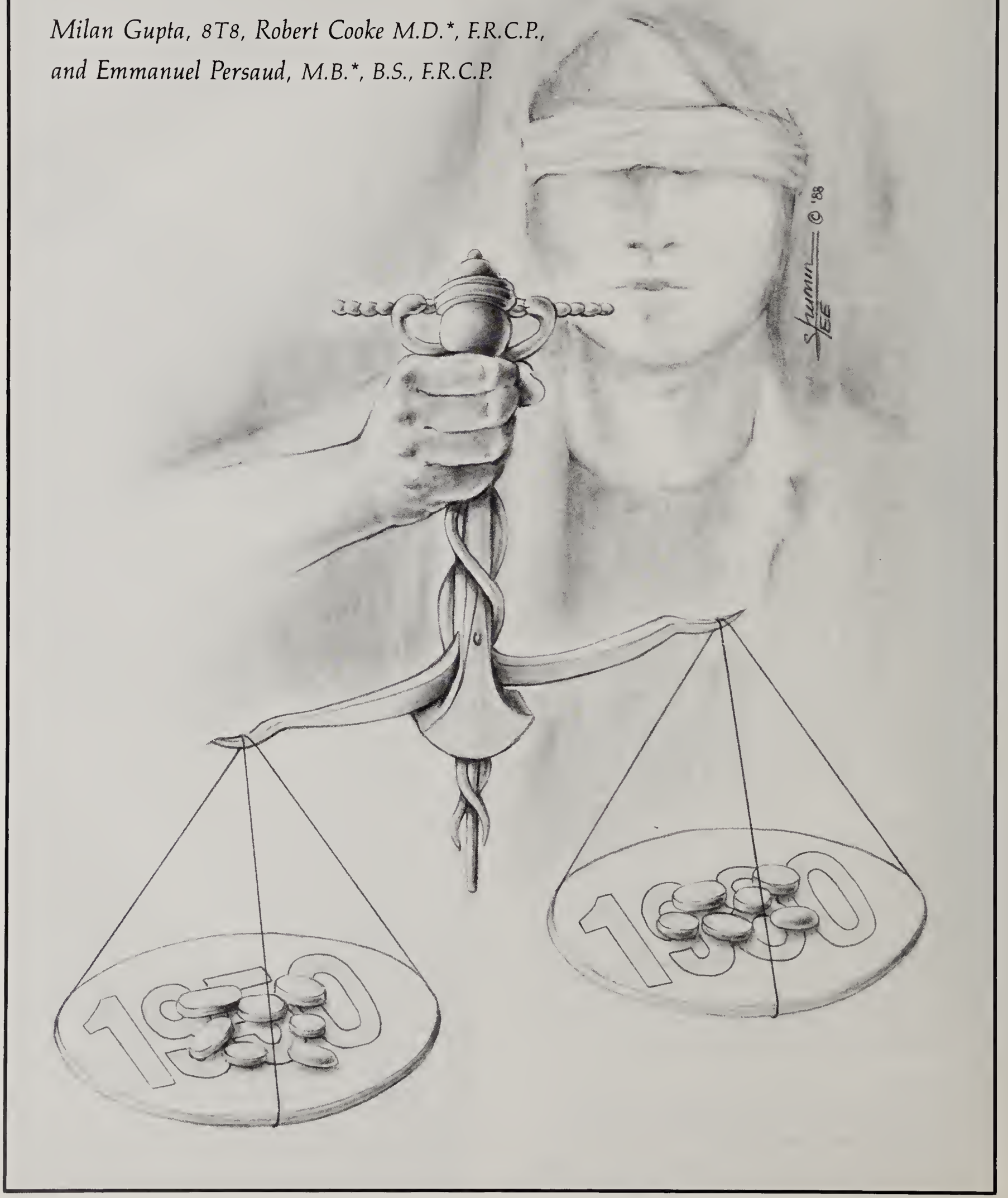


Anti-depressants and the Generation Gap

Milan Gupta, 8T8, Robert Cooke M.D., F.R.C.P.,*
and Emmanuel Persaud, M.B., B.S., F.R.C.P.*



PHARMACOLOGY

Abstract

Although the original tricyclic antidepressants introduced in the 1950's and 1960's remain in widespread use today, their undesirable side effects still render them unsuitable in many cases. This has led to the development of a new generation of compounds, reported to have similar efficacies but fewer adverse effects. Is there really a generation gap between conventional and newer antidepressants? Three recently developed drugs: amoxapine, maprotiline, and trazodone, offer little that is new. Alprazolam, a benzodiazepine, seems to be effective as an antidepressant, but still remains to be approved for clinical use as such. This review concludes that the newly developed drugs have no overwhelming advantages over older antidepressants.

Introduction

The introduction of tricyclic antidepressants and monoamine oxidase inhibitors thirty years ago represented an important advance in the understanding and treatment of depressive illness. The clinical efficacy of first generation drugs such as imipramine and amitriptyline has been repeatedly tested and confirmed by researchers world-wide. However, it has recently become apparent that these drugs can have incomplete efficacy and side effects that render them inadequate in many cases.[1]

Comparable or superior clinical efficacy, earlier onset of action, and reduced toxicity are the main criteria in the search for newer antidepressants.[2] Recently, several drugs have been released for clinical use, apparently having met these criteria. If this is so, then these new antidepressants represent a true advance in the treatment of depression. The problem of drug contraindication due to accompanying somatic conditions might be reduced, and patient compliance might increase. In the long run, a broader spectrum of patients, possibly even those previously resistant to standard drugs, could benefit.

Amoxapine

Amoxapine belongs to a new group of tricyclics, the dibenzooxazepines, and has been shown to be effective as an antidepressive agent. It is structurally and pharmacologically similar to imipramine, and is the demethylated derivative of loxapine, a neuroleptic with no apparent antidepressant activity.[3]

Amoxapine strongly inhibits the neuronal reuptake of norepinephrine.[4] It also inhibits serotonin reuptake, but to a much lesser extent. Amoxapine also blocks post-synaptic dopamine receptors, but has no effect upon monoamine oxidase.

Amoxapine has a pharmacologic half-life of 8 hours, and is biotransformed into two active metabolites. The major metabolite, 8-hydroxyamoxapine, has a long half-life of 30 hours. It is a potent blocker of norepinephrine reuptake, and has stronger serotonergic effects than its parent compound. A second metabolite, 7-hydroxyamoxapine, makes up a much smaller proportion and has a half-life of 4 hours. This metabolite, like amoxapine, blocks dopamine receptors, possibly explaining the extrapyramidal side effects occasionally seen in patients on this drug.[5] Thus, amoxapine administration must be monitored carefully for acute extrapyramidal symptoms.

The results of many double-blind placebo-controlled trials have shown that amoxapine is superior to placebo, with therapeutic efficacy similar to imipramine[6,7] and amitriptyline.[8] The dosage range for the older tricyclics is usually 150-300 mg/day, whereas the range for amoxapine is 300-600 mg/day.

Early reports suggested that amoxapine had a more rapid onset of action than other tricyclics,[9,4] with signs of improvement observed within one week of starting treatment. Lederle Laboratories, the manufacturers of amoxapine, claim that therapeutic effects may be seen as early as 4-7 days, and within two weeks in 80% of responders. However, both conventional tricyclics and amoxapine demonstrate similar effects in the third week.[10,11] An in-depth review by Lydiard et al. (1984)[3] suggests that amoxapine may show earlier effects, but the authors refused to make any definite conclusions. Further well-designed studies are recommended before any decisions can be made.

Some of the side effects of treatment with amoxapine resemble those of standard tricyclics, particularly imipramine. Sedation and anticholinergic effects are the two most common with amoxapine, with severity comparable to imipramine. It seems, then, that amoxapine fails to offer any advantages over presently-used tricyclics in this very important respect.

The cardiovascular effects of amoxapine in therapeutic doses are also similar to those of imipramine, occurring frequently but with little clinical significance.[12]. However, when comparing cases of amoxapine and imipramine overdoses, fewer serious cardiovascular effects have been noted with the former.[13,4].

Of great concern is the possibility of tardive dyskinesia, a disease involving severe motor dysfunction. Several cases have been reported with long-term treatment with amoxapine.[14] A second consideration, though of less concern, is the increased possibility of acute extrapyramidal symptoms such as dystonias, akathisia, and pseudo-parkinsonism.[15] This has been attributed to the fact that both amoxapine and its 7-hydroxy metabolite have an antagonistic action at dopamine receptors. Such dopamine blockade may also be in-

Acknowledgements

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volved in cases of hyperprolactinemia and galactorrhea which, like the acute extrapyramidal symptoms, are reversible upon cessation of treatment with amoxapine.[12] Such neuroendocrine effects are rarely seen with imipramine, and are, in fact, not common with amoxapine.

Maprotiline

Maprotiline belongs to a class of compounds known as the dibenzodicyclo-octadienes, and is structurally referred to as a tetracyclic. Pharmacologically, maprotiline is a specific reuptake inhibitor of norepinephrine. Unlike most other antidepressants, it has no effect upon serotonin metabolism, *in vitro*. It has been suggested therefore, that maprotiline may be especially beneficial to patients with suspected norepinephrine imbalances.[16] Maprotiline has a relatively long half-life of 40-50 hours. Numerous metabolites are formed in the liver, the principal one being desmethylmaprotiline. The dosage range for maprotiline is 150-300 mg/day.[16]

Maprotiline has been evaluated in cases of endogenous and neurotic depressions, and has been proven effective in both.[17] Recent research has shown maprotiline to be as effective as imipramine,[10,18] but claims that maprotiline may surpass imipramine in efficacy remain unsubstantiated.

Several investigators have suggested an earlier onset of action with maprotiline. As with most new antidepressants, many studies have been done, but few conclusions reached. Lydiard et al.[3] fail to find any significant difference in onset of action between imipramine and maprotiline, but admit that minor early differences are seen in favour of the newer drug.

Maprotiline has a slightly reduced side-effect profile, in that it has fewer atropine-like effects. Although patients still complain of the usual anticholinergic symptoms, such as blurred vision, constipation, dry mouth, and urinary retention, these are reported less frequently and in lower numbers. Sedation is more evident, possibly as a consequence of maprotiline's antihistaminic activity, and the incidence of skin rashes is also higher than for some other drugs.[9] Cardiovascular problems are reduced, with fewer cases of sinus tachycardia and postural hypotension.

Although all antidepressants lower the convulsive threshold to a certain degree, numerous reports of seizures with maprotiline therapy have raised concern. It was reported that 42% of all drug-induced convulsions in Britain, as of November, 1979, were due to maprotiline.[19] Other antidepressants accounted for only 28%. This figure, however, cannot be fully interpreted since the relative use of maprotiline in comparison to other antidepressants during this time is not known. It seems though, that maprotiline does considerably lower the seizure threshold at high doses, and possibly even at therapeutic doses.[14] Others argue that maprotiline is less likely to induce convulsions than tricyclics.[20] At present, then, the issue remains unresolved.

Trazodone

Trazodone is a phenylpiperazine derivative of triazolopyridine, the first such compound to be used in the treatment of depressive disorders.[10] It is perhaps unique among the approved antidepressants available today, differing from the others in pharmacological action, structure, and toxicity. First synthesized in 1966, and after 15 years of research and development, trazodone is now widely recognized as a valuable addition to the growing number of currently available antidepressants.

Trazodone's antidepressant action has been attributed to its role as a serotonin agonist. It selectively inhibits serotonin reuptake, potentiating it centrally. In contrast to the tricyclics, trazodone does not affect central norepinephrine reuptake, but may enhance its release.[21] Trazodone also seems to potentiate dopamine, but this property is still questionable.[22]

Trazodone has a half-life of 7-13 hours. It is metabolized via hydroxylation and conjugation, but the major form in plasma is unmetabolized trazodone. Its metabolites appear predominantly in the urine, where trazodone itself is found in minute amounts.[23] Treatment with trazodone requires 200-500 mg/day.

Trazodone has been shown to be definitely superior to placebo,[24,10] with comparable efficacy to imipramine.[25] Trazodone has also been proven effective in cases of neurotic depression with signs of associated anxiety.[24] Research suggests an earlier effect with trazodone, the first signs of improvement noticeable within 7 days,[26,27] whereas tricyclics require 2-3 weeks.[2]

Trazodone has an almost complete lack of anticholinergic activity, giving it a very high therapeutic index. In numerous clinical trials, the incidence of anticholinergic effects is similar in both trazodone and placebo-controlled patients,[27,23] and higher in patients on imipramine.[29] Trazodone's efficacy and safety data indicate a definite potential in geriatric cases.[30]

Sedation is the most common side effect associated with trazodone, with other mild effects including skin rashes, headaches, numbness and tingling.[31] Postural hypotension and dizziness have been noted, but few other cardiovascular effects have been reported.[22] Trazodone has essentially no effect upon heart rate, even in high doses.[23]

Although the side-effect profile seems promising so far, trazodone has been implicated in at least 11 cases of priapism with the first two weeks of treatment.[14] In five of these cases, surgical procedures were necessary,[32] with almost half resulting in permanent impotence.[33] Even though other adverse effects are rare, the presence of such unexpected complications as priapism indicates that further research with this drug is required.

Alprazolam

In an extensive literature review, Schatzberg and Cole (1978)[34] concluded that benzodiazepines, classically prescribed as anxiolytics, were not effective as antidepressants. Alprazolam, a triazolobenzodiazepine, seems to be a unique member of this group of drugs, since it has shown antidepressant activity. Although still not approved for use as an antidepressant, alprazolam may represent a safer and better-tolerated compound than

conventional tricyclics.[35]

Research indicates the existence of specific benzodiazepine receptors in localized areas of the brain.[36] It has been suggested that alprazolam binds to these receptors to exert its effect as an antianxiety agent.[37] Long-term treatment with reserpine in rats leads to an increase in the number of central beta-adrenoceptors, resulting in reserpine-induced depression.[38] Pretreatment with alprazolam (or imipramine) in reserpine-treated rats prevents this increase, suggesting a possible explanation for alprazolam's antidepressive effects.[39] Alprazolam does not affect norepinephrine or serotonin metabolism *in vitro*.

Alprazolam has a half-life of 10-12 hours, and is metabolized to 4-hydroxyalprazolam, alpha-hydroxyalprazolam, and alpha, 4-dihydroxyalprazolam.[40] Ongoing trials confirm alprazolam's potential in cases of depression with associated anxiety. Whether alprazolam is equally effective in cases of depression alone remains to be determined. Clinical trials have used doses up to 4 mg/day.

When effective, alprazolam is a rapidly-acting drug, with noticeable improvements by the end of the first week.[28,41] Alprazolam demonstrates side effects typical of other benzodiazepines, including drowsiness, headaches and nausea. Also included is the dangerous possibility of dependency and tolerance with long-term treatment, a well known problem associated with benzodiazepines. Alprazolam causes few anticholinergic effects, rendering it useful in the elderly. Other adverse effects are uncommon, ranking alprazolam among the safer compounds available for use today. Like most benzodiazepines, alprazolam has a low index of cardiotoxicity, and a low potential for lethality upon overdose.

Conclusion

It is obvious, therefore, that the clinician has a wide choice of antidepressant drugs to choose from, ranging from tricyclics and MAOIs, to newer nontricyclic antidepressants and benzodiazepines. However, it seems that the newer drugs may have been over-rated in their initial evaluation. Although they may offer some slight advantages, they are little better than already existing antidepressants, and offer little in the way of advances in treatment. Furthermore, until these new drugs are extensively tested and in widespread use, there remains the alarming possibility of unexpected serious adverse effects. In conclusion, then, there seems to be no real 'generation gap' when comparing first and second generation antidepressants. Research, though, is continuing to contribute to our present knowledge of the mechanisms involved in depression, and thus is increasing the forms of treatment available.

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Warnings: Massive acetaminophen overdose can be toxic and potentially fatal. In adults, hepatotoxicity from acetaminophen is unlikely to occur with overdoses of less than 10 grams ingested at one time and fatalities are unlikely to occur with overdoses of less than 15 grams ingested at one time.

This product contains codeine and should not be administered to children except on the advice of a physician.

Precautions: The sedative effect of codeine is additive to that of other CNS depressants, including alcohol. Dosage may need to be reduced when administering this product with other CNS depressants and patients should be cautioned about driving a car or operating potentially hazardous machinery if they become drowsy or show impaired mental or physical abilities while taking codeine.

This product has the potential for being abused.

Safety in pregnancy has not been established. The product should not be administered during pregnancy unless, in the judgment of the physician, the potential benefits outweigh the possible hazards.

Adverse effects: Acetaminophen may occasionally cause gastric upset. The incidence and severity of this adverse efect is less than that after A.S.A. administration.

The most common adverse effects of codeine include lightheadedness, drowsiness, nausea and constipation. Respiratory depression is seen in higher dosage.

Overdose symptoms: The manifestation of an overdose of TYLENOL NO. 1 FORTE acetaminophen with codeine capsules/tablets are those of acetaminophen overdose combined with an exaggeration of the adverse effects of codeine.

The early symptoms of acetaminophen overdose overlap the symptoms of codeine overdose and include gastrointestinal irritability, nausea, vomiting, anorexia, diaphoresis and pallor. Symptoms of hepatic necrosis may become evident from three to five days following ingestion.

In sufficient overdose codeine can cause euphoria, dysphoria, miosis, a decrease in respiratory rate, cyanosis, and hypotension. Death due to respiratory failure may result.

Treatment: The stomach should be emptied promptly by lavage or induction of emesis with syrup of ipecac.

The hepatotoxic effect of acetaminophen overdose can be countered with the antidote N-acetylcysteine. Further information on the clinical course of acetaminophen overdose and its treatment with N-acetylcysteine is available from McNEIL Consumer Products Company, McNeil Pharmaceutical (Canada) Ltd. or the nearest Poison Control Centre.

The respiratory depressant effect of codeine overdose can be countered with a specific narcotic antagonist such as naloxone. In the presence of hypoventilation or apnea, oxygen should be administered and respiration assisted or controlled. A patent airway must be maintained.

Adult dosage: TYLENOL NO. 1 FORTE acetaminophen with codeine: 1 or 2 capsules/tablets 3 – 4 times daily as required, not to exceed 8 capsules/tablets in a 24-hour period. Do not exceed the recommended dose unless advised by a physician. Use longer than 5 days only on the advice of a physician.

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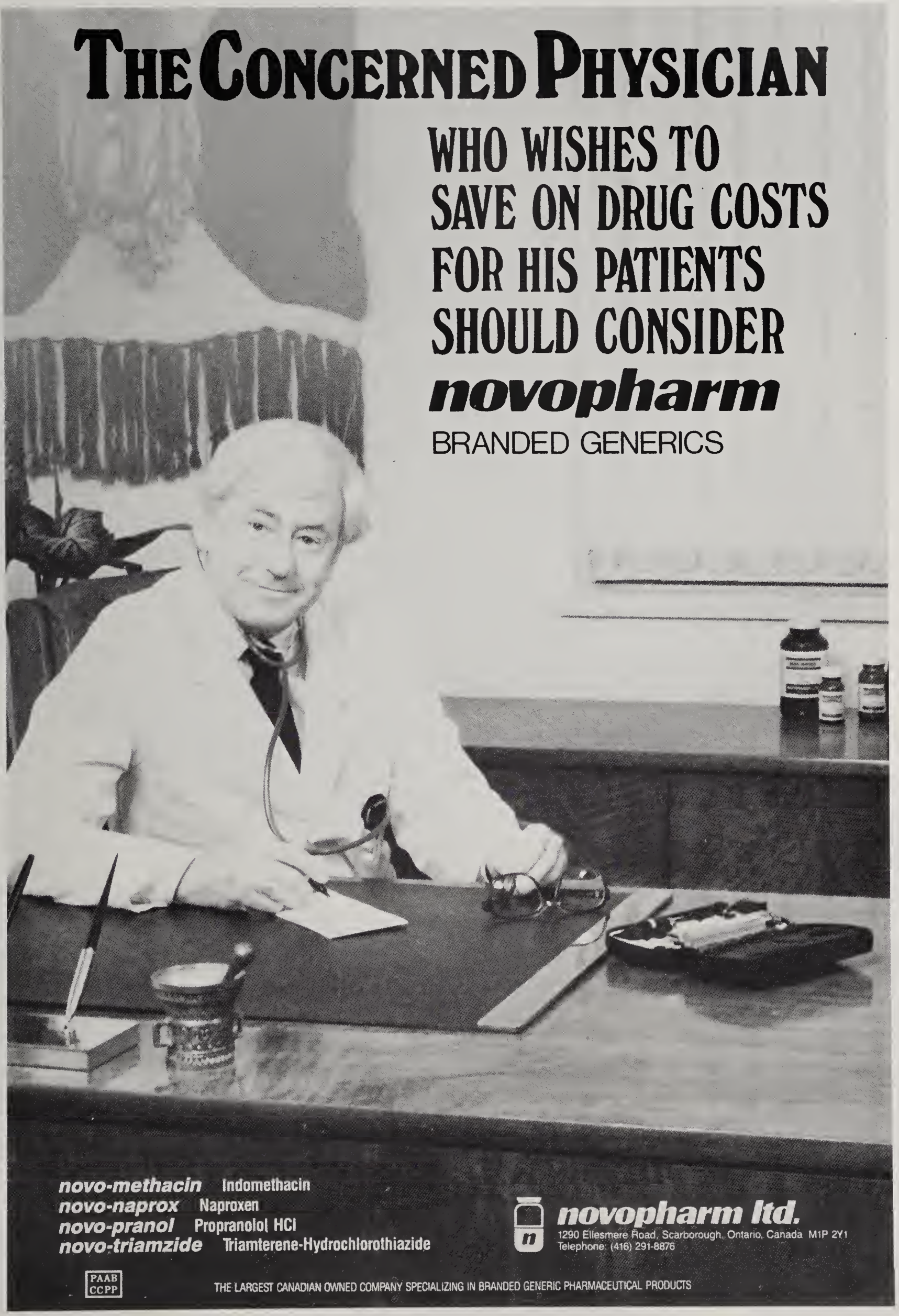

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Obsessive-Compulsive Disorder: *Rituals and Ruminations of Mind*

Michael Paré, 8T8, Sturla E. Bruun-Meyer, M.B., B.Ch., F.R.C.P.(C), M.R.C. Psych., and Salvatore Mallia, M.D.*

Abstract

The authors briefly outline the diagnosis, etiology, epidemiology, as well as the common presentations and treatment of obsessive-compulsive disorder. Several clinical vignettes are used to illustrate the various syndromes and unique symptomatology of this psychopathological condition.

Introduction

Case 1. Consider this patient with ruminations: A 25-year-old man had recurrent thoughts of killing his younger brother by cutting off his head. He struggled and resisted these thoughts and impulses, first by avoiding sharp objects, then avoiding his brother, and finally by avoiding his entire family. Eventually he found it necessary to avoid using the letters of the alphabet that made up his brother's initials.[1]

Case 2. Consider this patient with rituals: A 32-year-old man employed as an assembly line worker in an electronics firm developed the ritual where, before being able to do his soldering, he had to tap the work bench three times with his left hand, then stamp three times on the floor with his left foot, followed by three stamps with his right foot. At first this unusual behaviour did not overly interfere with his work, but merely slowed him down. The problem progressed, however, when he began to doubt whether or not he had correctly completed a sequence of tapping manoeuvers: "Did I really do it right? Am I sure I tapped three times? Did I stamp with my left foot first?" He began to repeat the entire tapping procedure over again in order to allay his fears that he had initially performed it incorrectly, but his doubts remained. Eventually, he was forced to leave his job since almost his entire working day was taken up by these rituals.[2]

Some of the dramatic characteristics of obsessive-compulsive disorders can be seen in the above clinical vignettes. An "obsession", as in Case 1, is defined as any recurrent intrusive thought (rumination), feeling, image, or impulse that invades consciousness, is ego-alien, unwanted and cannot be removed by logical effort. "Compulsions" refers to recurrent, "forced", unwanted stereotyped behaviours, often ritualistic, which the patient may regard as repugnant. The daily paroxysms of tapping in Case 2 highlight some of these features.

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Obsessive-Compulsive phenomena are not restricted to any one mental disorder but rather are found in a variety of psychiatric conditions, including Obsessive-Compulsive Disorder (OCD), affective disorders, schizophrenia, organic brain syndromes, Tourette's syndrome, anorexia nervosa, and, indeed, are also found in normal mental life.

The obsessions and compulsions associated with OCD have certain features.[1,2,6]

I) The idea or impulse intrudes forcefully and persistently into the person's awareness.

II) A powerful feeling of dread may accompany the idea or impulse. Sometimes countermeasures, either psychological or behavioural, are mounted, against the initial idea or impulse, as in the following case:

Case 3. This patient, a young man, had the obsessive thought: "My father will die", whenever he turned off a light. He thus became compelled to touch the switch again and think the countermeasure: "I take back that thought", leading him to believe that he was counteracting any effect from his first obsessive thought.[2]

III) The obsessions and compulsions are experienced as ego-alien (not a usual part of one's self experience); ego-dystonic (unwanted and unacceptable) and involuntarily produced (uncontrollable). Nevertheless they are recognized and experienced as internal in origin, in contrast with delusions, which are ideas attributed to an external source.[3]

IV) The obsessions and compulsions are recognized as senseless and irrational. Again this distinguishes them from delusions where the patient has lost insight and so does not appreciate the irrationality of the thoughts.

Case 4. "Mr. C., a 20-year-old college student, was brought to the hospital by his parents because his rituals prevented him from pursuing his undergraduate studies in physics". Because of a "... preoccupation with contamination ... he showered ... several times a day..." "He recognized that his contamination idea was irrational, stating: 'That's what bothers me the most. I am such a

rational person, it's very upsetting to have such an irrational idea.' ".[3]

V) The individual suffering from obsessions and compulsions describes an internal struggle or resistance against these preoccupations. Patients will say: "No matter how hard I try, I can't get this crazy thought out of my mind."

Diagnosis

To conform to the current official American Psychiatric Association diagnosis of Obsessive-Compulsive Disorder (OCD) certain criteria contained in the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III) must be fulfilled (see Table I). The obsessions and compulsions must cause significant interference in the patient's social or role functioning and must not be a secondary phenomenon associated with another mental illness. They may, however, often have other secondary symptoms such as depression (the most common), anxiety, aggression, phobias, sleep disturbances, sexual dysfunctions and appetite disorders.

It is particularly important to distinguish OCD from obsessive-compulsive character traits. People having an "obsessional character" (the so-called "anal personality") exhibit behavioural phenomena characterized by a preoccupation with sexuality, aggression and control. They are unemotional, perfectionistic and excessively devoted to work. Although in the extreme this can be classified as a compulsive character disorder, the mere presence of obsessional character traits is not in itself an indication of any abnormality. On the contrary, many of society's most productive and successful people (including medical students and physicians) are considerably obsessional. Differentiation of OCD from obsessive-compulsive character traits (and compulsive character disorder) can be made when one remembers that persons with OCD have ego-dystonic, ego-alien symptoms, whereas obsessive-compulsive character traits are ego-syntonic, usually do not provoke resistance, nor a sense of compulsion.

Etiology

The etiologic basis of OCD remains obscure. Psychoanalytic formulations suggest that the symptom presentation is a derivative of an unconscious wish or fear and the defense against it. The symptom may distract the patient from unacceptable ideas which it is feared could otherwise emerge. Mechanisms such as isolation, undoing reaction formation, regression and magical thinking are involved in containing the anxiety (affect) but fail. A "false connection" is made between the affect and other ideas, which then turn into obsessive ideas. Psychoanalytic theories have been criticized for generating untestable hypotheses.

Neurobiological studies have not fared much better, however. Anatomically such areas as the temporal lobes and cingulum in the limbic system have been suggested as being involved. Possible nonsuppression of the dexamethasone suppression test and decreased rapid eye movement latency during sleep in these patients has not led to any clearer understanding of a specific neurological dysfunction in this disorder. There may, how-

Table 1
Diagnostic criteria for Obsessive Compulsive Disorder

A. Either obsessions or compulsions:

Obsessions: recurrent, persistent ideas, thoughts, images, or impulses that are ego-dystonic, i.e., they are not experienced as voluntarily produced, but rather as thoughts that invade consciousness and are experienced as senseless or repugnant. Attempts are made to ignore or suppress them.

Compulsions: repetitive and seemingly purposeful behaviors that are performed according to certain rules or in a stereotyped fashion. The behavior is not an end in itself, but is designed to produce or prevent some future event or situation. However, either the activity is not connected in a realistic way with what it is designed to produce or prevent, or may be clearly excessive. The act is performed with a sense of subjective compulsion coupled with a desire to resist the compulsion (at least initially). The individual generally recognizes the senselessness of the behavior (this may not be true for young children) and does not derive pleasure from carrying out the activity, although it provides a release of tension.

B. The obsessions or compulsions are a significant source of distress to the individual or interfere with social or role functioning.

C. Not due to another mental disorder, such as Tourette's Disorder, Schizophrenia, Major Depression, or Organic Mental Disorder.

Table 2
Common Syndromes of Obsessive-Compulsive Disorder*

Obsession	Compulsion	Resistance	% of OCD Cases
Contamination	Washing Rituals	Moderate	≃ 50%
Doubt	Checking Rituals	High	≃ 20%
Pure Obsessional (usually sexual or aggressive)	None	Compulsive	≃ 25%
Primary Obsessional slowness	None	Low	Very rare

(*Modified from Insel, 1985[1]).

KAREN VISSER

ever, be a genetic predisposition to acquire OCD which, when coupled with particular family styles of behaviour, could lead to the disorder developing at times of environmental or psychological stress.

Epidemiology

Obsessive-Compulsive disorder is a chronic, waxing and waning psychiatric condition. Most develop during adolescence or the early 20s; only 10% after 35 years. The disorder is more common in females than in males, the ratio approaching 2:1. Recent evidence suggests that the prevalence of OCD may be as high as 1-2% of the general population.[5] This, however is not reflected in the number of cases seen in the psychiatric clinic since patients with this disorder tend to be secretive about their symptoms and avoid disclosing them to their physicians. The lifetime prevalence is 2-3%. The clear distinction between OCD and obsessive-compulsive character traits is supported by epidemiologic evidence that reveals that compulsive character features are neither necessary or sufficient for the development of OCD. Thus being "obsessional" does not lead to OCD, a fact that will undoubtably be of comfort to many medical students.

Common Syndromes

Although OCD may present in many ways, the great majority of cases are found to exhibit a rather limited range of symptoms (see Table 2).

The largest group is made up of persons who are obsessed with the possibility of contamination. They are so irrationally afraid of such things as germs, disease, dirt and poisons, that they may engage in washing rituals lasting up to several hours per day.

Case 5. "Mr. A., a married 57-year-old aerospace mechanic became concerned about bits of metal or glass on his skin. He began checking and washing compulsively although he recognized that this behaviour was bizarre and unreasonable. He also expressed fear that the particles on his skin might contaminate others and cause injury". Eventually the problem progressed to a point where "he stood naked for hours in the middle of his room complaining that he was unable to move because he would contaminate others".[3] Occasionally this overwhelming fear of contamination is so great there is a deliberate avoidance of cleaning and personal hygiene, a seemingly paradoxical situation. This symptomatology is particularly evident in the case of Howard Hughes, the famous American billionaire who developed OCD in adulthood.

Case 6. "A fear of germs was predominant since childhood. By age 54, in 1959, his dread of contamination had reached such proportions that he resided in houses and hotels sealed off from the outside world by elaborate security systems. In his own room, the drapes over the sealed windows were taped shut with masking tape so that he was kept in darkness 24 hours a day. He allowed no human contact and insisted that an article he had to handle be "insulated" with up to 100 sheets of tissue. When his aids brought him food or documents to sign, they would use "paddles", also constructed of numerous layers of tissue, to avoid touching these articles that would later come in contact with Hughes. He would not permit his room to be cleaned nor would he allow his bedding to be changed, but rather added layer upon layer of "insulation" consisting of paper towelling. Eating and waste elimination were surrounded by such elaborate and exhaustive rituals in an effort to maintain a germ-free environment that he developed extremely poor dietary habits, chronic constipation and occasional faecal and urinary incontinence... ultimately, his fears and compulsions, his refusal to take food or water, and massive drug abuse (to intravenous codeine) led to death from dehydration and renal failure. His physical condition, filthy and unkempt, was reported to be worse than that of the most decrepit skid row derelict. Paradoxically, the compulsive behaviours presumably adopted to reduce his fear of death contributed directly to it".[6]

Another common presentation of OCD may be termed "psychopathological doubt", where the overwhelming concerns may only temporarily, if at all, be diminished by the compulsion of checking.

Case 7. A man in his thirties fearing that he would push a stranger off the subway platform was compelled to keep his arms and hands glued rigidly to his sides. Despite his strict precautions he became obsessed with the fear that he had, after all, inadvertently knocked someone off the subway platform. He struggled with himself for weeks to dispel what he rationally knew was a foolish notion, but was eventually compelled to call the transport authority to check so as to reassure himself that there had not, in fact, been any such accident.[2]

A third relatively common form of OCD is a purely obsessional state, characterized by recurrent, intrusive thoughts, with no associated compulsive behaviour. The most common ruminations are of an aggressive or sexual nature. For example, a young mother was constantly obsessed with the fear that she would strangle her six-month old baby. Another young woman of 23-years of age was shocked one day when the mental image flashed in her mind of her father and herself undressing one another and joining in a sexual embrace. Rather than being a fleeting image it constantly reoccurred leading to marked distress.

Much less commonly found is the syndrome of "primary obsessional slowness". These patients are so perfectionistic and concerned about the tiniest detail about carrying out an action that they are virtually paralysed. A patient may present with stasis pigmentation of both ankles resulting from the several hours spent every day in front of the bathroom sink and mirror, doing daily routines.

It is important to stress that although the above four presentations of OCD do occur in a pure form, overlap between the various syndromes occurs more commonly and patients may even shift from one form to another over time.

Treatment

As no simple remedies are available for this complex and difficult psychiatric condition, it is clearly best to approach the patient with a multi-dimensional treatment plan. Supportive psychotherapy can be useful in relieving distress, monitoring progress, helping the family and in maximizing compliance with other therapeutic modalities. Although psychodynamic insight-oriented treatments have generally been found to be less successful, behavioural approaches involving exposure ("flooding") and thought stopping techniques have fared much better, especially with milder symptoms. Psychosurgery (cingulotomy) is of help in some severely disabled chronic patients.

A wide variety of medications have been tried in OCD, with the most consistently effective drug therapy being antidepressants such as phenelzine, imipramine and clomipramine (Anafranil). Double-blind studies have shown that these are useful in reducing symptoms in OCD, with clomipramine being the more effective. Their effect is probably by a mechanism distinct from their antidepressant action and involving more than serotonin reuptake blockade alone.

Although useful in diminishing the symptoms of OCD, without the presence of depression, it remains unclear whether these particular antidepressants can be regarded as having a specific antiobsessional action, although this remains a fascinating possibility and a promising starting point for future psychopharmacological research. They are also a major new direction in providing help for sufferers of a major chronic medical problem.

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Vascularized Fibular Grafting

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*Robin R. Richards, M.D., F.R.C.S.(C)**

Abstract

Vascularized fibular grafting for the treatment of non-traumatic osteonecrosis of the femoral head is a valuable new technique for the management of this difficult orthopaedic problem. This procedure permits retention of the femoral head and the preservation of joint function in patients in the early stages of the disease.

Introduction

Osteonecrosis of the femoral head is a disorder characterized by pain and limitation of motion in the affected hip. A wide variety of conditions are related to the development of this disabling disorder (Table 1.)[1] The causes of osteonecrosis of the femoral head can be divided into two groups: traumatic and non-traumatic. Traumatic causes include fracture of the femoral neck, dislocation of the hip, and slipped capital femoral epiphysis. In these conditions there is disruption of the blood supply to the head of the femur. The second category encompasses a large number of conditions in which the exact cause of the necrosis remains uncertain, although evidence suggests vascular involvement here as well. The incidence of non-traumatic osteonecrosis of the femoral head is significantly higher in males than in females (with a male:female ratio of 4:1 being reported) and there is bilateral involvement of the hips in approximately 40% to 70% of cases.[2,3,4] Excessive alcohol consumption[3,4,5] and corticosteroid use[4,5,6] are significant risk factors in the occurrence of this disease. Indeed, Solomon[7] has reported that in a study of 57 patients with the disorder, a history of either alcohol abuse or corticosteroid use was obtained in every case.

The treatment of osteonecrosis of the femoral head has included: (i) prolonged non-weight bearing in an attempt to prevent further collapse of the femoral head while repair and revascularization occur; (ii) core decompression to relieve the elevated venous pressure within the femoral head; (iii) bone grafting to hasten the repair process; (iv) osteotomy to alter the weight-bearing surface of the hip to prevent further collapse, aid repair, and minimize degenerative changes; and (v) arthroplasty. Treatment of femoral head necrosis with a vascularized fibular graft is a relatively new development that offers patients the benefits of a living bone graft. In this paper we discuss the use of this technique in the management of a patient with non-traumatic osteonecrosis of the femoral head.

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Operative Technique

Two teams work simultaneously, one harvesting the vascularized fibular graft and the other preparing the hip to receive the graft.

Elevation of Fibular Graft: A longitudinal lateral incision is made in the leg of the affected limb and the common peroneal nerve is identified. The interval between the peronei and the posterior musculature is developed and the fibular diaphysis is exposed. The popliteal, anterior and posterior tibial, and peroneal arteries are identified. A 15 cm section of the fibular diaphysis is then measured and two osteotomies are made. The peroneal artery is identified in the distal portion of the wound and divided. The fibula is then dissected preserving the muscular cuff surrounding the vascular pedicle. The peroneal artery and its vena comitantes are preserved. The tourniquet is then deflated. Profuse bleeding from the fibular graft should be observed indicating patency of vasculature.

Hip Preparation: A longitudinal lateral incision is made over the hip and the lateral circumflex artery is identified. The lateral portion of the femur is exposed, the cortex is perforated, and a guide wire is passed into the anterosuperior femoral head into the segment of osteonecrotic bone. Reamers are used to create a tract in the femoral neck and head to receive the fibular graft. Curettes are used to remove the osteonecrotic bone. A cancellous bone graft is then taken from the greater trochanter and packed up into the zenith of the femoral head leaving a space for the fibular graft.

Transfer of Graft from Leg to Hip: The fibular graft is removed from the leg and this wound is closed. The anterior cortex of the femur at the insertion point of the graft is removed in order to prevent compression of the vascular pedicle. The graft is inserted and impacted into the femoral head and a wire is used to hold the graft firmly in position.

An anastomosis is carried out between the peroneal artery and the lateral femoral circumflex artery in an end-to-end fashion using an operating microscope (Figure 1A, 1B). Upon removal of the vascular clamps the peroneal artery should be observed to pulsate and the fibular graft should bleed if the anastomosis is patent. The wound is then thoroughly irrigated and the

Table 1
Conditions Associated with the Development of Avascular Necrosis of the Femoral Head[1]

Fractures of the femoral neck
Traumatic hip dislocation
Slipped capital femoral epiphysis
Following intracapsular osteotomy of the hip
Sickle cell anemia
Other anemias
Alcohol abuse
Corticosteroid administration
Rheumatoid arthritis
Systemic lupus erythematosis
Chronic pancreatitis
Occlusive vascular disease
Osteomyelitis
Radiation
Gaucher's disease
Renal tranplantation
Caisson disease
Pregnancy
Tumors
Others: Leriche syndrome
Coagulation deficiencies
Cretinoid epiphyseal dysgenesis
Dyschondroplasia
Charcot's arthropathy
Idiopathic

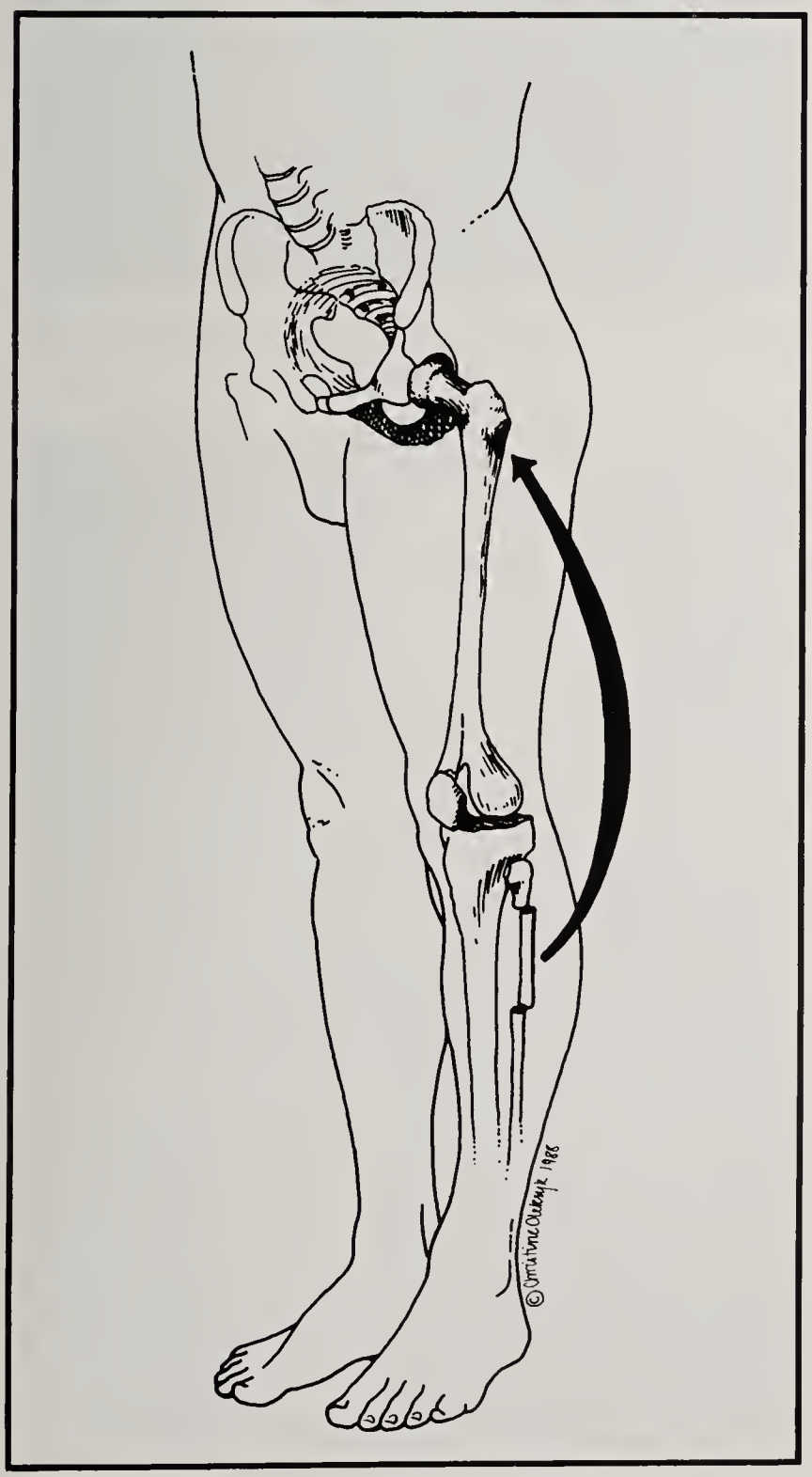

Figure 1A: Diagram showing the transfer of the fibular graft with its attached vessels from the leg to the hip.

fascia is closed over the fibula. Following closure of the wound a dressing is applied to the limb and a posterior slab is used for the leg. The patients have a bone scan twenty-four hours postoperatively to confirm that the fibular graft is perfused. The patients are mobilized forty-eight hours postoperatively, non-weight bearing on crutches.

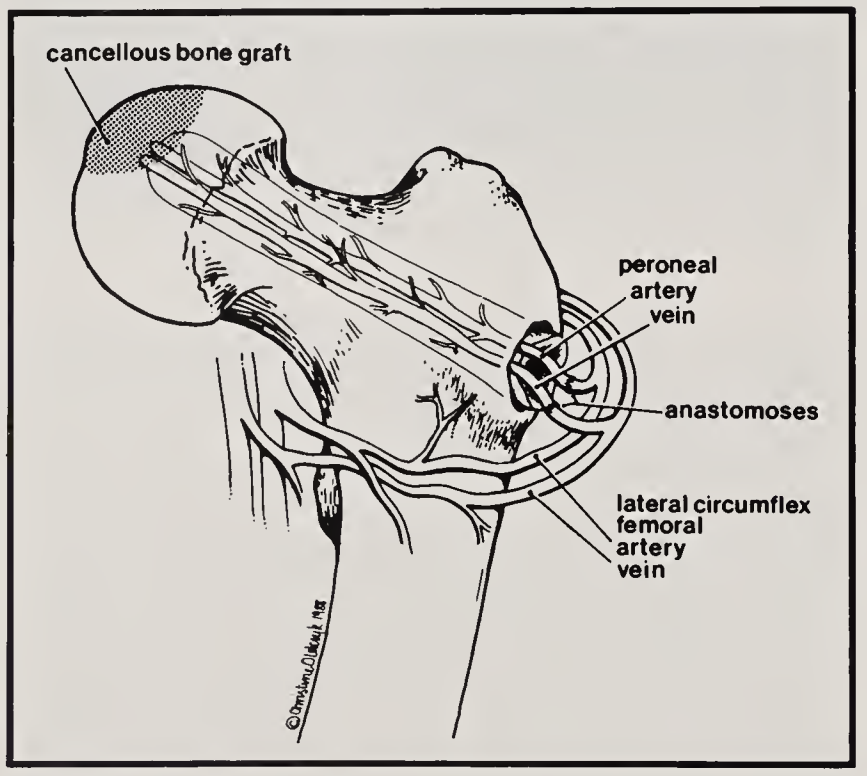

Figure 1B: Figure 1B shows the vascularized fibular graft in place. The graft is inserted into the anterior segment of the femoral head. Osteonecrosis most frequently involves the anterosuperior portion of the femoral head and an image intensifier is used to be certain that the vascularized graft is in place in this area.

Case Report

The patient is a 38 year old woman with systemic lupus erythematosis who developed mild aching discomfort in her left hip in early 1985. She had been treated with Prednisone for two years prior to the onset of the pain. A radiograph of her left hip in December 1985 revealed segmental sclerosis of the femoral head with slight irregularity of the joint surface but no gross collapse (Figure 2). Although the

Figure 2: A/P radiograph of patient's left hip. There is segmental sclerosis of the superior portion of the femoral head with slight irregularity of the joint surface.

patient had been non-weight bearing for almost one year when initially seen, the pain had gradually worsened. Hip motion was good with only slight limitation of internal rotation.

Radiographs showed segmental necrosis of the left femoral head with slight collapse. A tomogram confirmed the diagnosis of segmental osteonecrosis in the anterior-superior portion of the femoral head (Figure 3). An angiogram demonstrated suitable vasculature for a free fibular graft and the operation was performed in October 1986 (Figure 4). A bone scan forty-eight hours postoperatively revealed improved vascularization of the left femoral head (Figure 5).

The patient was mobilized non-weightbearing on crutches for six months following which weightbearing as tolerated was permitted. At follow-up one year later, she had a full range of motion in the hip and was pain free. Radiographs revealed no further collapse of the joint space, with the sphericity of the femoral head having been maintained. The vascularized fibular graft was well healed (Figure 6).

Discussion

The natural history of non-traumatic osteonecrosis of the femoral head is one of progressive joint destruction. Most femoral heads, if untreated, will collapse.

Figure 3: A tomogram of the femoral head confirms a segment of osteonecrosis with slight collapse of the joint surface. The segment involved is, typically, in the anterosuperior portion of the femoral head.

Figure 4: Postoperative A/P radiograph shows vascularized fibular graft inserted into the superior portion of the femoral head.

Figure 5: Postoperative bone scan shows the fibular graft to be well vascularized and to have increased the uptake of the femoral head.

This results in degenerative arthrosis producing severe hip dysfunction and disability.[2,3,8] Attempts at non-surgical management of the hip such as protected non-weight bearing have generally been unsuccessful.[9] Hence, surgical treatment is indicated in symptomatic patients in an attempt to arrest the progression of the disorder and preserve hip function.

Ficat[10] has suggested that the underlying mechanism in the pathogenesis of the disorder is an obstruction of the osseous microcirculation with intramedullary stasis. This leads to increasing pressure within the confines of the femoral head, metabolic disturbance, and finally anoxia and necrosis. Hence, treatment of the disorder by decompression has been advocated by several authors who have achieved good results with this technique.[7,10,11] However, a recent study of forty core decompression procedures for osteonecrosis of the femoral head by Camp and Colwell[12] led them to conclude that the procedure is ineffective (60%.of patients who had undergone decompression before collapse of the femoral head had occurred experienced progression of the disorder either clinically and/or radiologically) and that it has a significant morbidity because of the complication of fracture, (there was a 12% incidence of major fractures associated with the procedure in the study).

Non-vascularized bone grafts have also been used in the treatment of non-traumatic osteonecrosis of the femoral head. Traditionally, bone grafting has entailed the insertion of devascularized cortical tibial bone grafts through the base of the greater trochanter and femoral neck into the head of the femur as described by Phemister in 1949 for the treatment of traumatic osteonecrosis of the femoral head.[13] Application of this technique for the treatment of non-traumatic osteonecrosis of the femoral head has given mixed results. Boettcher et al,[9] have reported that the insertion of bone grafts into the femoral head gave excellent results

Figure 6: A/P radiograph at one-year follow-up shows vascularized fibular graft to be well healed and reveals no further collapse of joint surface.

and Bonfiglio and Voke[14] concluded that this procedure was so effective in restoring viability to the necrotic femoral head (in almost 80% of patients) as to justify its continued use in the management of osteonecrosis of the femoral head. However, Marcus et al,[15] in reviewing 53 patients, the majority of whom were treated with Phemister type bone grafts, noted that the results of this procedure were usually disappointing and that the natural progression of the disease appeared to be unaffected. Smith et al,[16] points out that proper placement of the graft into the subchondral cortex of the anterior superior quadrant of the femoral head is critical to the success of this technique.

The main purpose of non-vascularized bone grafts is to provide mechanical support for the subchondral bone and articular cartilage while revascularization of the head occurs; however, such grafts remain necrotic for years following implantation.[17] Given its necrotic status, the avascular bone graft is unable to remodel until it is revascularized. The result is that the unsupported articular cartilage and subchondral bone continue to bear much of the weight so that collapse of the head and joint destruction ultimately occur. The advantage of the vascularized fibular graft is that it is living and hence has the ability to remodel and hypertrophy in response to the new stresses placed on it.[18]

The use of vascularized fibular grafts for the treatment of osteonecrosis of the femoral head has been described by Judet et al[19] who recommended its use in young patients with advanced osteonecrosis of the femoral head in whom the only alternative would have been total joint replacement. Given the limitation of function imposed by hip arthrodesis and the frequent failure of total hip replacement in young patients[20] vascularized fibular grafting offers such individuals the choice of avoiding the finality of a total joint arthroplasty while retaining the comfortable function of their natural hip joint. Fujimaki and Yamauchi[21] reported complete relief of symptoms and no progression of necrosis or deformity in three out of four young patients with early non-traumatic osteonecrosis of the femoral head who were treated with the use of this technique. There was persistence of mild pain, bony absorption, and some progression of the deformity in the fourth patient. Although the long-term outcome of this method of treatment remains to be seen, the early results appear promising.

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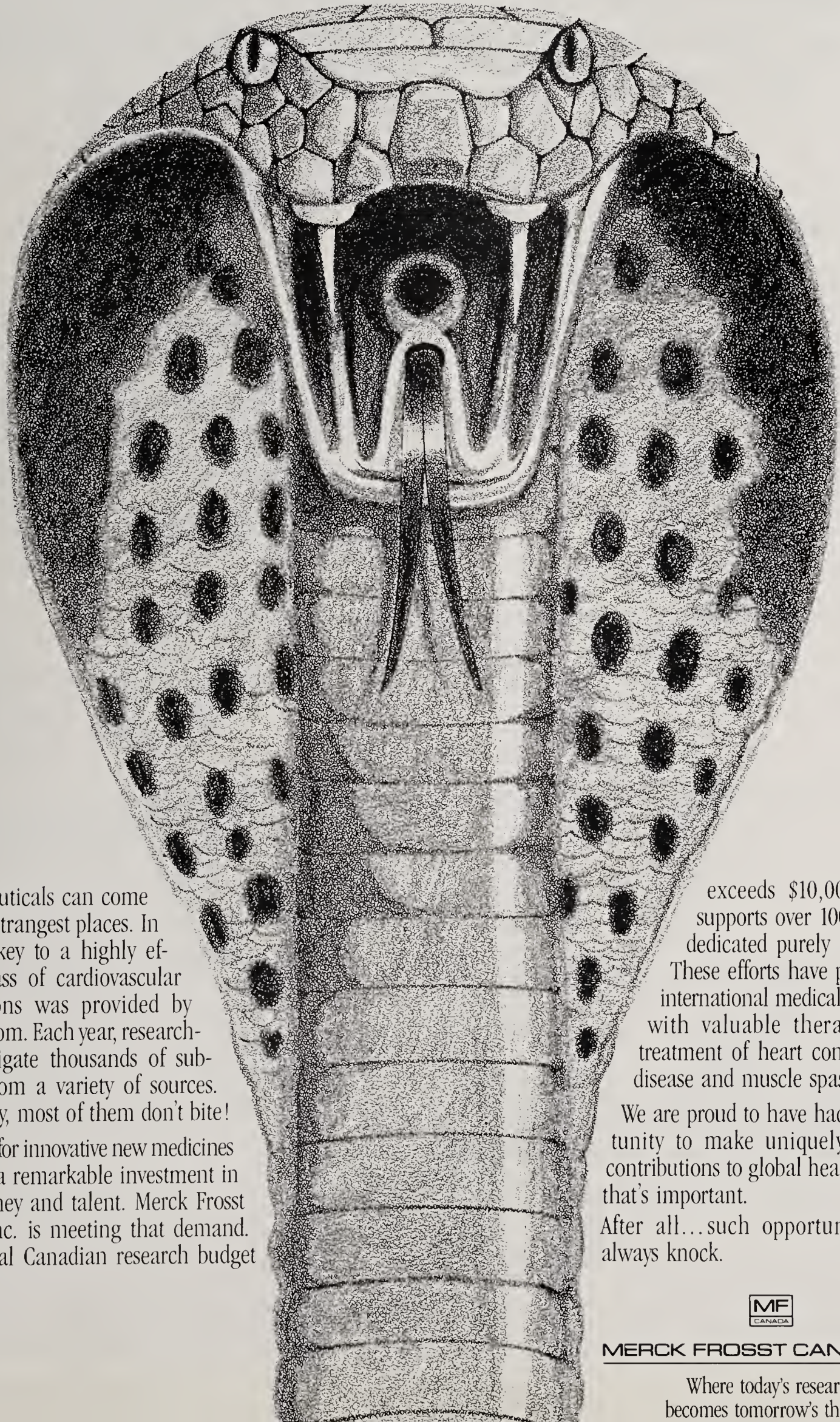

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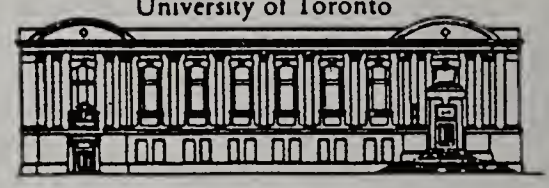

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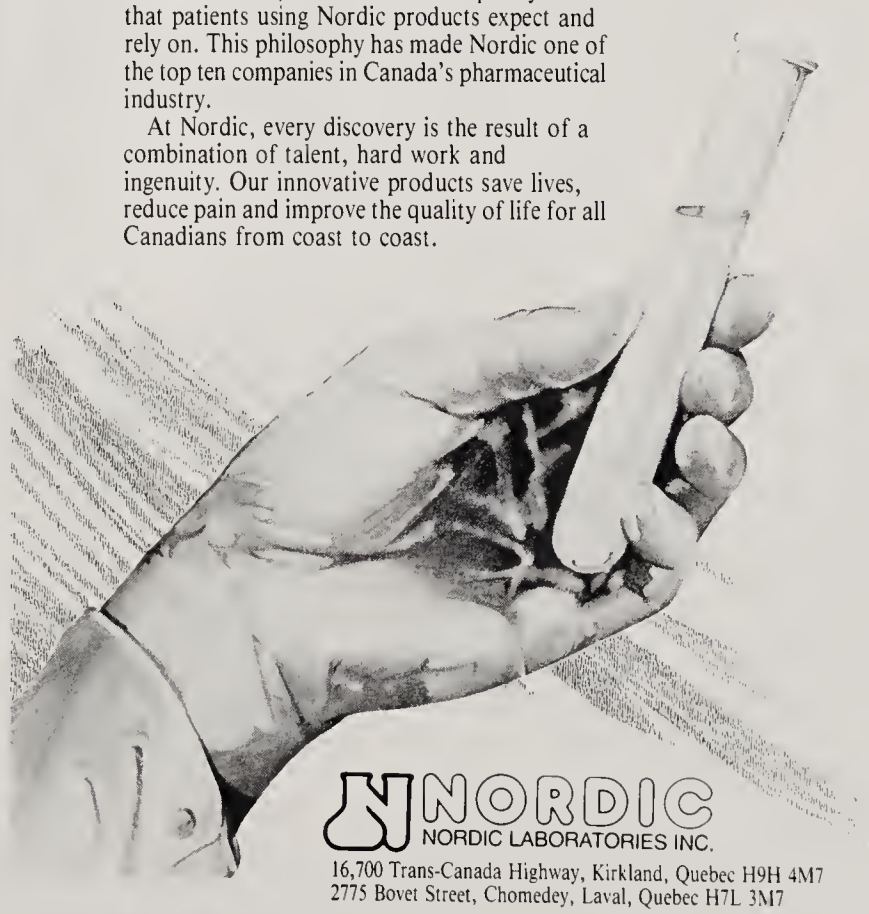

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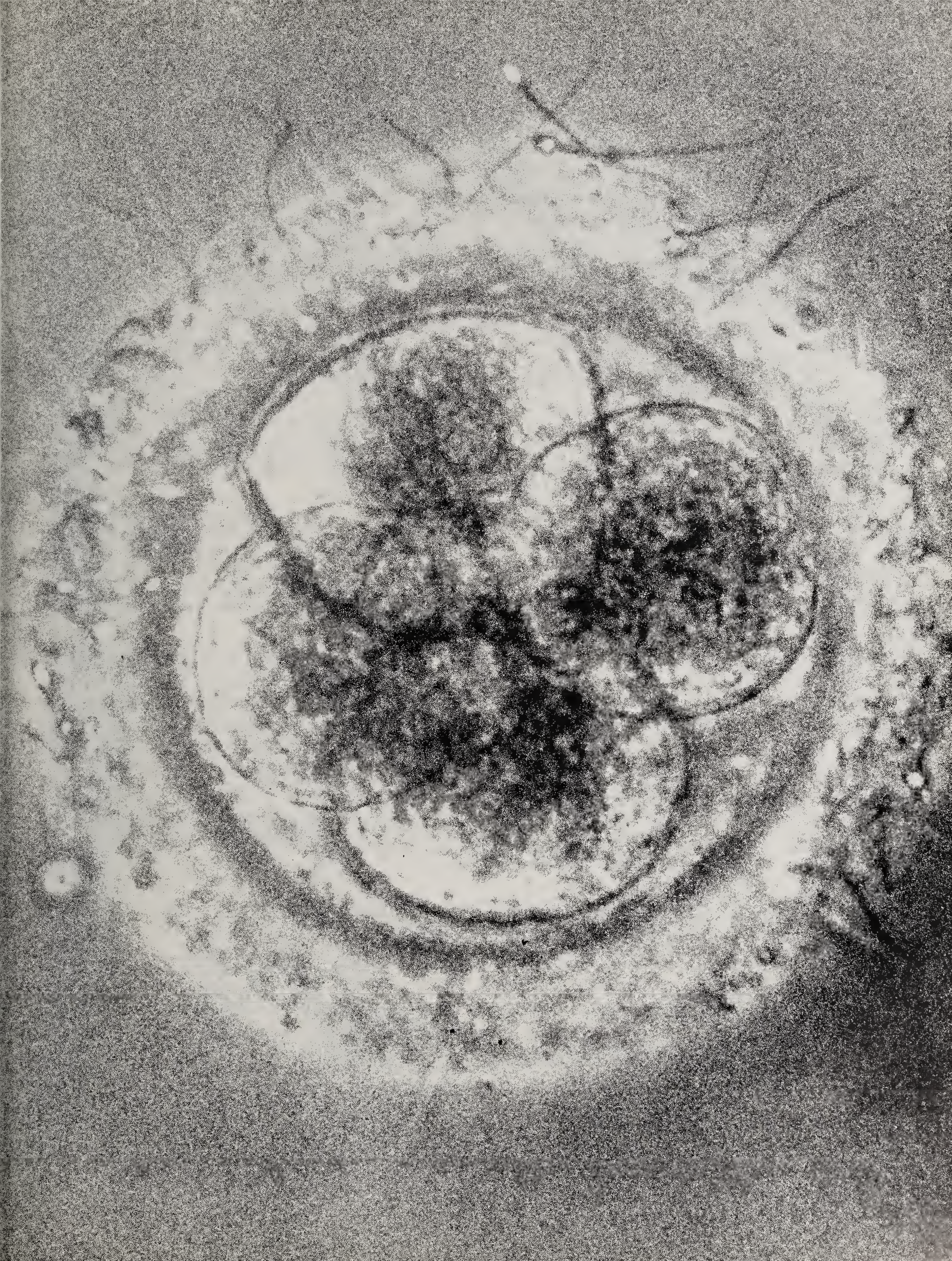

The Elderly Gravida: *A Pregnancy at Risk?*

Susan Zeldin, 8T8

Abstract

The number of women over the age of 35 who are bearing children is increasing. This paper discusses the evidence that increasing maternal age places the mother and foetus at greater risk during pregnancy and labour. It appears that the increased dangers should not be sufficient to deter an older woman from deciding to become pregnant.

Introduction

A recent study has shown that in the last decade there has been a 37% increase in the number of women over the age of 35 who are giving birth for the first time.[3] This trend is likely to continue in the foreseeable future as more and more women join the work force and decide to establish themselves in their careers or professions before raising a family and, thus, postponing motherhood. It is because of this increasing trend to bear children in later years that it is necessary to establish the extent to which elderly gravidas should be considered as pregnancies at risk. An elderly gravida is defined as a pregnant woman who is 35 years or older at the time of delivery.

This paper will address this problem by discussing some of the risks associated with the elderly gravida that have been documented in the literature. The risks may be divided into three groups:

1. Maternal Risks - risks to the mother's health during pregnancy.
2. Foetal Risks - birth defects and congenital abnormalities associated with chromosomal abnormalities.
3. Risks Associated with Labor and Delivery.

Maternal Risks

The literature does not necessarily point to a direct link between advanced maternal age and risks to the mother's health. It does, however, suggest that an indirect relationship exists in as much as advanced maternal age is a predisposing factor to certain medical conditions. Most of the maternal risks attributed to pregnancy such as diabetes, kidney disorders, cardiovascular problems, and hypertension are associated with pre-existing medical disorders which are more prevalent in older women.[3]

Toxemia, for example, occurs more frequently in elderly gravidas than in younger gravidas.[6] This increased frequency, however, can be explained by the fact that hypertension is more prevalent in older individuals and that hypertension itself is one of the major predisposing factors to the development of pre-eclampsia during pregnancy.

Further, pre-eclampsia, if detected early, can be treated under proper medical supervision and need not necessarily be a risk factor in elderly gravidas. Accordingly, provided that the mother is in good general health, there is no evidence to suggest that she is at greater risk than a younger mother in this regard.

Foetal Risks

There is, on the other hand, evidence that advanced maternal age does produce a significantly increased risk to the unborn foetus. The risks associated with elderly gravidas are those of spontaneous abortion and birth defects associated with chromosomal abnormalities.[10]

The increased risk of spontaneous abortion associated with older women is evident in the results of a study performed in 1979. This study concluded that

spontaneous abortions in the first trimester of pregnancy occurred 3½ times more frequently among women over 35 than in women aged 20 to 29.[3] Statistics also show that elderly gravidas are at a higher risk for stillbirths.[3]

It is also pertinent to note that 40-60% of the spontaneous abortions occurring during the first trimester can be attributed to chromosomal abnormalities which have a definite correlation to advanced maternal age.[3] The most notable and perhaps well studied chromosomal abnormality associated with older women is trisomy 21 or Down's syndrome. The rate of trisomy 21 increases from 1 in 2000 live births at maternal age 20 to 1 in 280 at age 35 and increases further to 1 in 100 at age 40.[3] The specific incidences of trisomy 18 and 13 as well as those of Klinefelter's syndrome (XXY) and XXX syndrome increase exponentially with advancing maternal age.[4]

All these trisomies have been linked to the result of non-disjunction in the maternal first meiotic division. In fact, 79% of Down's syndrome births to elderly gravidas can be attributed to non-disjunction.[4] The correlation between increased incidence of chromosomal non-disjunction and maternal age can easily be understood when one considers the length of time the oocyte remains in the ovary. All the primary oocytes are

formed during foetal life and remain in a suspended prophase of the first meiotic division until ovulation. Therefore, in the case of an elderly gravida, the oocyte at the time of ovulation is more than 35 years old.[1] During this prolonged period, harmful effects of the environment on the spindle fibers formed during metaphase may lead to abnormal segregation of the chromosomes and hence, non-disjunction. This phenomenon is less frequent in the second meiotic division since the period between ovulation and fertilization is much shorter.

The effect of maternal age on the risks of other birth defects, such as central nervous system malformations, cleft lip and palate, and congenital heart defects is not as clearly defined. It has been postulated that the frequency of these disorders may be due to changes in the gametes, possible increased permeability of the placenta to teratogens and changes in the intrauterine environment associated with age.[11] These conclusions, however, are speculative and warrant further study.

A positive correlation was found between elderly gravidas and the development of diabetes mellitus among their children.[11] The study concluded that there was an increased prevalence of diabetes among children born to older women, notwithstanding the fact that none of the elderly gravidas in this study were diabetics themselves.

Risks Associated with Labor and Delivery

The risks of the elderly gravida with respect to labor are less well defined and are still under investigation. Recent literature has concluded that advanced maternal age has little significant effect on the overall duration of normal labor. Some studies suggest an increased incidence of protracted and prolonged labor with

advanced age. This may be associated with decreased efficiency of the myometrium of the uterus with age, but other factors such as increased use of anesthesia and analgesia have also been implicated.[2]

The risk of abnormal labor progression also increases with age. Specifically, there may be a prolongation of either the first or second stage of labor, both of which lead to an increased risk of perinatal death.[6] This abnormal progression may also lead to malposition of the foetus which in turn may result in complications of delivery.[8] A study by Freedman et al. (1965) demonstrated an increase of secondary arrest of dilation in gravidas over the age of 35.[6] This finding was further supported in another study, performed in 1980, in which a significant correlation was found between age and arrest disorders. In fact, the incidence was more than twice as great among women over 35 than of those under the age of 20.[2]

Discussion

In summary, the evidence seems to suggest that there is an increased incidence in maternal and foetal risks in older women. These disorders, however, are not necessarily different with respect to etiology and prognosis from those that occur in younger gravidas. Because of the increased potential for abnormalities, however, physicians should be made aware that elderly gravidas do require more careful monitoring during pregnancy, especially in the presence of preexisting predisposing factors. If this is kept in mind, and with the judicious use of recent advances in medical technology such as amniocentesis, foetal monitoring and ultrasound, the risks of the elderly gravida can be greatly reduced.

In fact, according to Dr. Mansfield, professor of nursing at Pennsylvania State University, "maternal age is shown to be irrelevant in predicting most pregnancy outcomes when other factors, themselves bearing an association with age, are separated statistically from the age variable. Women who are healthy and have had no gynecologic complications in the past, should feel confident about having a baby after the age of 35."[7]

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Patient with Night Cough, Polycythemia, Cyanosis, and Previous Heart Surgery

Murari Patodia B.Sc. 8T8, Robert H. Hyland, M.D. F.R.C.P.(C), John R. Ross, M.D., F.R.C.P.(C), Robin R. Gray, M.D., F.R.C.P.(C)
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Case History

A 47 year old Greek male presented to the Emergency Department complaining of a non-productive nocturnal cough of one weeks duration. The patient denied any fever, chest pain or dyspnea. A detailed history was difficult because the patient spoke little English. It was, however, learned that he had had heart surgery more than once and that he is phlebotomized on a regular basis (every 2-3 months).

Physical examination revealed a thin man in no apparent distress with mild central cyanosis, and significant clubbing of fingers and toes. His heart rate was 75 beats/minute and regular with an occasional premature beat. Blood pressure in the right arm was 95/70 sitting. Respiratory rate was 18 breaths/minute. His chest was

Figure 1a and b: Chest PA and lateral views. Arrow indicates rib notching.

clear, but had multiple surgical scars anteriorly. Except for certain cardiovascular findings the rest of the examination was unremarkable. The cardiovascular findings were as follows:

1. arterial collaterals and bruits over the left clavicle
2. a grade 2/6 pansystolic murmer at the lower left sternal border without obvious respiratory variation.
3. normal intensity S1, but a single S2, no S3 or S4.
4. no radial or brachial pulse in the left arm.

His CBC showed a hemoglobin of 164 g/L and a hematocrit of 0.54. The chest x-ray (Figure 1a and b) and EKG (Figure 2) are below. What is your diagnosis?

The Answer

The diagnosis is partial repair of tetralogy of Fallot. A study of this case demonstrates many of the significant features of tetralogy of Fallot (TOF), but presented first is some background information on this condition.

Although first described in 1671 by Stensen, this tetrad of congenital heart defects bears the name of Fallot, who in 1888 completed the description of this condition in a series of patients.[1] TOF is the most common cyanosing congenital heart defect (CCHD) in the adult. It accounts for approximately 10% of the 8-10/1000 live births that have congenital heart defects.[2] The following is a list of the components of the tetralogy in decreasing hemodynamic importance:

1. Right ventricular outflow tract obstruction (ROTO)
2. Ventricular septal defect (VSD)
3. Right ventricular hypertrophy (RVH)
4. An aorta that straddles the ventricular septum.[1,3]

This patient was born in 1940 and was essentially asymptomatic until his early teens when he noticed that he fatigued easily and suffered from episodes of dyspnea and cyanosis which were relieved by assuming a squatting position.

This presentation demonstrates that cyanosing congenital heart defects may present much later than "blue babies". In fact these congenital defects may not become symptomatic until much later life, sometimes not until the 4th or 5th decade.[3] One reason for this is that the ROTO which may be mild or moderate at birth and hence asymptomatic, may become more severe with time. This is especially true of infundibular obstruction due to hypertrophy of the myocardium, which is the most common form of ROTO in TOF.[1] The obstruction can also be due to stenosis or atresia of the usually bicuspid pulmonary valve, pulmonary artery (PA) agenesis, PA coarctation or diffuse PA hypoplasia.[2]

The cyanosis that was noted was caused by a shunting of blood from the right ventricle to the left ventricle through the VSD. Initially this patient was cyanotic

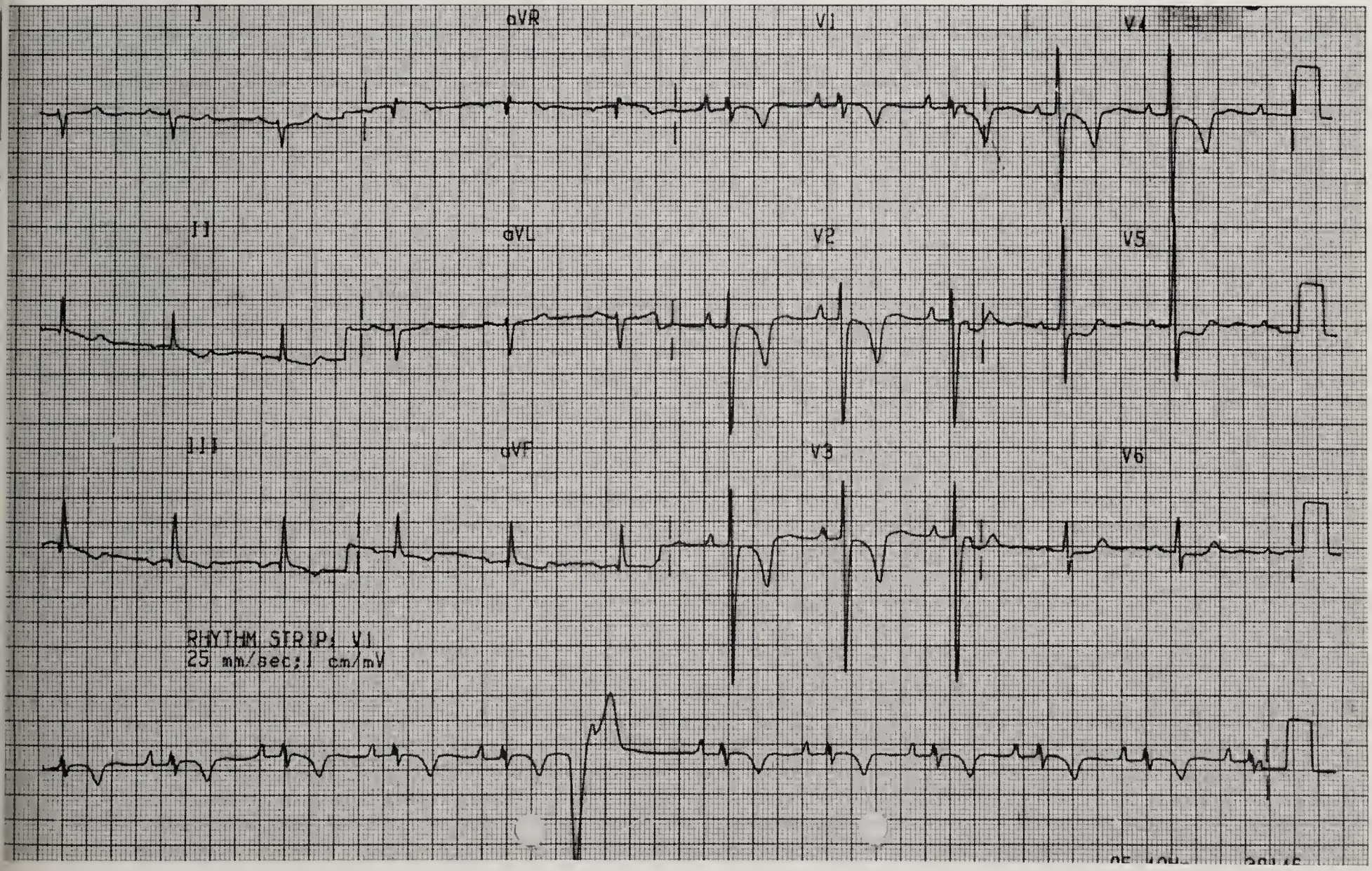

Figure 2: Electrocardiogram showing occasional Ventricular Premature Beat, poor QRS progression and t-wave inversion in the anterior leads.

only on exertion. With the increasing venous return to the right heart during exertion, the ROTO becomes restrictive to flow. The right heart, in an attempt to overcome the obstruction, generates higher pressures and becomes hypertrophied. When the pulmonary pressure reaches systemic levels, the VSD, which as a rule is large (the size of the aortic orifice), acts as a release valve and right to left shunting occurs.[1]

Squatting, a behavior demonstrated by this patient, is characteristic of CCHD. There are several mechanisms by which this relieves the symptoms. Firstly, squatting increases systemic vascular resistance. This results in higher pressures in the left ventricle and leads to a left to right shunt which, in turn, increases pulmonary blood flow. As well, squatting decreases venous return so that a smaller volume of unsaturated blood (which occurs after exercise) reaches the heart and ultimately the systemic circulation. Finally, squatting is believed to trap some of the oxygen depleted venous blood in the legs.[3]

By 1960, the symptoms had become overwhelming for the now 20 year old patient. At this time, he had the first of 3 palliative surgical procedures. First, as developed in 1944 by Blalock and Taussig, a left subclavian artery to left pulmonary artery anastomosis was performed to increase the pulmonary blood flow and hence increase the amount of oxygenated blood getting to the systemic circulation.[4]

This systemic to pulmonary shunt which is known as a Blalock-Taussig shunt (B-T) accounts for many of his cardiovascular and radiologic findings. His left radial pulse was absent because the arm was being supplied by collaterals, some of which crossed the left clavicle, thus accounting for the bruit. Another supply of collaterals developed from the ipsilateral intercostal arteries. These hypertrophied collaterals produce the characteristic rib notching on the left side that is seen by the arrow on the chest x-ray.[1,5] (Figure 1) These changes can be prevented or reduced if a modified B-T is performed using a synthetic conduit between the two arteries, and thus preserving the subclavian artery.[1] The increase in the blood flow through the pulmonary vasculature on the same side as the shunt as compared to the contralateral side results in more prominent vascular markings on the ipsilateral side as noted in the chest x-ray.[1,5] (Figure 1)

After this operation the patient was completely asymptomatic for 2-3 years. Gradually over the next four years his symptoms returned. His exercise tolerance was reduced to 100-200 meters at a slow pace on level ground. At this point he was referred to the National Institute of Health (Bethesda) for investigation and treatment.

The return of symptoms after a B-T can be due to one of two reasons. The first occurs more commonly when a B-T is performed in an infant because many B-T's fail to grow as the child does. However, the more common cause is that the shunt occludes either suddenly or gradually.[3]

During the investigations at NIH it was determined that the B-T had occluded. A complete intracardiac repair was planned at the time. The first complete repair was successfully performed by Lillehei in 1956.[1] The essentials of a TOF repair involves right ventriculotomy, closure of the VSD with a synthetic patch, release of the ROTO through either a patching of the outflow tract or resection of the infundibulum and a pulmonary valve repair if necessary.[1,6]

A Brock procedure (R ventricular infundibulectomy and pulmonary valvotomy) was performed. Repair of the VSD could not be performed because the right coronary artery crossed the right ventricular outflow tract and was at risk. This anomaly has been noted to occur in 4% of all TOF.[1] Other anomalies that have been associated with TOF are right sided aortic arch (20%), persistent left superior vena cava, aortic valve insufficiency, endocardial cushion defect, tricuspid atresia, and artrial septal defect.[1,2]

Ten days post-operatively the patient suffered a small stroke from a paradoxical embolus. This embolus, most likely due to a deep vein thrombosis which developed during the perioperative period, is one of the complications related to the polycythemia seen in TOF patients. The polycythemia is secondary to chronic cyanosis and can be treated with phlebotomy as is done with this patient.[3] Other complications of TOF include right bundle branch block, occurring most commonly in older patients, infective endocarditis, and cerebral abscess formation.[3]

The patient recovered and remained well so that he was not seen again until 1975. At that time he was admitted to hospital for hemoptysis. His symptomatology was mild to moderate. It was believed that blood flow through the pulmonary vasculature was decreasing, possibly due to pulmonary hypertension. As a result he was most probably developing collateral flow through his bronchial vasculature. The development of this collateral system can make initial diagnosis of TOF difficult because the patient may present with cyanosis, hemoptysis, and wheezing. The latter two symptoms are due to the dilated bronchial vasculature.[3] These collaterals also produce a continuous murmur in the chest (the systolic murmur heard in TOF is due to flow through the ROTO and not the VSD) and confound simple determination of patency of B-T shunts. Therefore to be sure a B-T shunt is functioning, angiography must be performed.[3,7]

In 1985 the patient had become markedly cyanotic. At this time it was determined that the ROTO was worsening and the B-T shunt was also completely occluded. In an attempt to increase the pulmonary blood flow, a palliative procedure (a right internal mammary artery to right pulmonary artery anastomosis) was performed. Since then, the patient has done well with only minor limitations.

It is difficult to assess long term prognosis of this patient. In patients with complete repairs, however, the prognosis is excellent with 75% living to the 4th or 5th decade as compared with only 5% survival to this point without surgery.[3,8] In one followup study of 106 complete repairs, it was found that patients could carry on with an essentially normal life, and even pregnancy could be very well tolerated.[9]

The key to the diagnosis of TOF is a high index of

suspicion, keeping in mind that the patient may present at any age. Some key clues are 1) paroxysmal cyanotic attacks, 2) history of squatting for relief, 3) wide pulse pressure (due to shunt), 4) S2 with a decreased or absent P component, 5) a systolic murmur at 2nd or 3rd intercostal space, 6) ECG findings indicative of RVH and 7) the following lung radiographic findings; decreased lung markings, normal cardiac size, uplift of the ventricular apex, decreased or absent pulmonary artery shadow and prominent bronchial arteries and right sided aortic arch (20%).[1,3,5,6]

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TRAINING IN RADIATION ONCOLOGY

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Peripheral Nerve Grafting

Dr. Alan R. Hudson, Professor and Chairman, Division of Neurosurgery, University of Toronto

Although the first clinical nerve graft was performed approximately 100 years ago, the techniques did not find favour until the mid-1960's at which time microneurosurgical techniques were introduced. A very large basic research and clinical experience has been accumulated by a Toronto group led by Dr. Alan Hudson of St. Michael's Hospital and Dr. Susan McKinnon of Sunnybrook Hospital. In the small and large animal experiments and in the human operating room autografts have been employed. The patient has to sacrifice the function of the donor peripheral nerve, usually in the leg, so that a more important function e.g. elbow flexion can be restored following nerve injury. In recent years this group have concentrated on the use of allografts and the modification of the immunologically mediated rejection response by the drug Cyclosporin. Both animal and primate experimental results are very promising and protocols are being prepared for the first series of human allograft experiments. Nerves will be harvested at the same time that multiple organ procurement is underway in the standard fashion. These allografts will be employed to overcome gaps in peripheral nerve tissue caused by previous injury. Much of the experimental work leading up to this clinical initiative has been performed by Dr. Jamie Bain, a Plastic Surgery resident, and the entire group consists of Neurosurgeons, Plastic Surgeons, Transplant physicians and basic scientists. The collaborative effort includes men and women of different professional disciplines situated in various hospitals of the University of Toronto system. Can allografts be used successfully at acceptable risk in human patients? All the available evidence points to an affirmative answer and it is anticipated that Toronto will continue to be at the forefront of innovation in patients suffering from severe peripheral nerve injuries.

Corticosteroids as Adjunctive Therapy of Pneumocystic Carinii Pneumonia in Patients with Acquired Immunodeficiency Syndrome (AIDS)

Tony D'Urzo, 9T0

The most common pulmonary infection associated with AIDS is pneumocystic carinii pneumonia (PCP). With convential therapy, patients who present with PCP in respiratory failure suffer a mortality rate approaching 90%. Recently, MacFadden et al. from the University of Toronto[1] treated 10 patients with PCP in progressive respiratory failure with conventional therapy (supplementary oxygen, intravenous fluids, nutritional support and wherever appropriate, ventilatory support) and the addition of methylprednilosone. Another group of 8 patients with comparable disease were treated solely with antibiotics and supportive therapy. Survival in the patients receiving methylprednilosone was 90% (9/10) as compared with 25% (2/8) in those patients who had not received adjunctive treatment. MacFadden et al.[1] also reported that treatment with corticosteroids resulted in rapid subjective improvement in symptoms, a decrease in the alveolar-arterial gradient for oxygen within 16 hours and the ablation of fever within 24 hours. While it is known that exogenous steroids exert analogous overlapping anti-inflammatory and anti-immune effects, the mechanism(s) underlying the observations reported by MacFadden et al.[1] have suggested that a reduction of pulmonary inflammation associated with corticosteroid therapy results in an improvement of respiratory function thereby allowing time for antibiotics to become effective. Although this report indicates that the addition of methylprednilosone may reduce the mortality rate of individuals with PCP and AIDS, these authors also stress that the indications for therapy should be clarified by a randomized blinded trial and that in no instance should steroids be used in the absence of accepted conventional therapy.

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Trauma Research at Sunnybrook Medical Centre

The National Institutes of Health (NIH), the funding agency for medical research of the United States federal government, has recently awarded a grant to the Trauma Unit of Sunnybrook Medical Centre of Toronto to carry out the project, "Motor Vehicle Accident Victims: Alcohol as a Factor in Injury and Recovery". This project, headed by Dr. R. McMurtry, director of Trauma and Emergency Services of Sunnybrook Medical Centre, is a collaboration between Sunnybrook Medical Centre and the Addiction Research Foundation. The goal is to look at survivors of motor vehicle accidents who are treated at Sunnybrook's Trauma Unit, and try to identify the characteristics of those drivers who are more likely than others to have bad and severe accidents. These investigators have already determined a positive correlation between alcohol consumption and severity of accident, and other preliminary results indicate that approximately 30% of the motor vehicle accident victims brought into Sunnybrook's Trauma Unit had been drinking. This project also aims at constructing a psycho-social profile of the high-risk driver, trying to get a picture of the victims' attitudes, personality, and personal history. Hence, the researchers working on this project utilize a multi-discipline approach to investigate all of the possible factors leading to severe accidents for this group of patients. In this way, prevention programs against road accidents can be designed accordingly. (Adapted from "Research News", Sunnybrook Medical Centre)

Post-Dysenteric Reactive Arthritis

Alfred Sem, 8T9

Reactive arthritis (ReA) refers to an aseptic synovitis which develops after an antecedent, extra-articular infection usually of either the gastrointestinal or genitourinary tracts. With respect to the host, certain class I HLA genes, or genes closely related thereto, appear to confer disease susceptibility. With respect to the pathogens, certain gram-negative bacteria notably *Yersinia*, *Salmonella*, *Shigella* and *Campylobacter* have the capability of provoking the arthritis. The pathogenesis of ReA remains obscure although the microbe-HLA relationship has suggested several hypotheses such as molecular mimicry of host and microbial antigens. These studies have frequently been hampered in the past by problems in defining the appropriate control groups. In addition, the incidence of ReA and the definition of clinical features and natural history have proved difficult to establish on the basis of isolated case reports. Previous reports generally estimate incidence on pooled populations over time following different outbreaks, and the arthritogenic potential, even among the same pathogen, may vary according to serotypes. A Toronto group, led by Dr. Inman of Toronto Western Hospital, overcame these problems by examining a large population which had been exposed to the same enteric pathogen at the same time. In September 1984, during the papal visit to Canada, a large number of police officers developed a diarrheal disease after convening at a single site to supervise a public event. Subsequent investigation traced the source to contaminated food in a prepackaged lunch provided to the officers, and identified the pathogen as *Salmonella typhimurim* phage 22. Among the 1608 individuals potentially exposed to the contaminated food, 473 met the case definition, and each was sent a questionnaire. Of those 340 who responded, 198 individuals had associated extra enteric features during the diarrheal illness, and 137 individuals of the latter agreed to participate in a follow-up study, including evaluation by family physicians and follow-up assessment by the physiotherapists of the Arthritis Society. Twenty-one individuals could not be located for assessment. Based on the statistical analysis done on the other 116 individuals with adequate data, this study defines the incidence of post-*Salmonella* ReA, describes the clinical features of the disease, and confirms the strong immunogenetic susceptibility linked to the B locus of HLA.

BOOK REVIEWS

Becoming a Doctor: A Journey of Initiation in Medical School

by Melvin Konner

Elizabeth Sifton Books-Viking, New York, 377 pages, $19.95

Melvin Konner was a thirty-three-year-old Harvard professor of anthropology, with two years of field work in the Kalahari desert and a well-received book on human nature behind him, when he applied and was accepted to medical school. These somewhat exceptional circumstances allowed him a more mature, contemplative approach to the exigencies of medical training, and while he himself is most keenly aware of his exceptionality, it is certainly evident in both his skilled writing technique and his thoughtful commentary. Unfortunately, though, important meta-clinical themes are often obscured by the personality excesses of the author.

For example, Konner packs enough elitist, self-righteous arrogance into his brief preface to **Becoming a Doctor** that even if he were to suppress all self-congratulation in the narrative itself (which he emphatically does not) he would still emerge looking like a decidedly pompous over-achiever. And yet, despite this serious weakness, the book is a thought-provoking essay, at once illuminating for the general reader and a "must-read" for any medical student looking for an ideological "second opinion".

Konner's "compensatory pause" (as he refers to the book with a metaphor borrowed from cardiology) is a specialty-by-specialty journal of his third-year clinical rotation. But it is as much a powerful critique and sociological analysis of modern medicine as it is a readable, often engrossing, collection of case histories. The author evokes some strong sympathy for his anti medical-establishment leanings without resorting to the shrill sensationalist tactics of other "medical heretics". He decries the decline of humanism in the pursuit of technology, but he offers many warm, inspiring anecdotes to show that all is *not* lost. At one point, he asks a sixth-year neurology resident one of the most difficult of all medical-relating questions: How do you tell a patient he is going to die? The doctor's ingenuous response is one of the best take-home messages the book has.

Because of his anthropological background, Konner is able to encompass a wide range of socially relevant issues with more than passing interest. These include areas as ethically challenging as D.N.R. (do not resuscitate) codes as well as more mundane yet pervasive behavioral dispositions such as alcoholism, homosexuality and even smoking.

Although he eventually completed a psychiatric residency and currently is affiliated academically with the Department of Psychiatry at Emory University, Konner has returned to anthropology with no plans to practice clinical medicine. His medical school experience thus has a George Plimpton-like quality, but his reluctance to pursue a medical career is almost a tacit expression of holier-than-thou hauteur. While he puts forth a strong and at times convincing argument against certain aspects of modern medical practice, Konner's sensitive volume intimately does more to celebrate the romance and wonder that is medicine.

Deena Ages 9T0

Doctors & Patients

by Dannie Abse, Oxford University Press, New York, 1984, 114 pages, $6.99.

This book is one in the series of "small Oxford books", which "are pocket anthologies on a wide variety of entertaining subjects, designed to amuse and illuminate through a choice selection of verse and prose and accompanied by delightful illustrations".

Five aspects of medicine are covered with anecdotal excerpts from literary works of fact (these tend to be autobiographies, biographies, and essays) and fiction (ranging from the plays of Molière to the prose of Pasternak), as well as poetry.

The book opens at the root of medical practice: the apprenticeship period of the future doctor. This is undoubtedly the most entertaining part of the book in that the anecdotes cover those facets of medical education to which every physician and student have been exposed in the course of his or her training. Reflections on the choice of medicine as a career are certainly examined. The relevance of teaching techniques, the initial impact of that crucial medical course of anatomy, the ridiculousness of certain exams, and the eccentricities of certain clinicians are cleverly depicted.

The next topic to be explored are doctors themselves — what makes a good doctor and what makes a bad doctor. This section discusses topics from the philosophies of ancient Greek physicians to the unscrupulous practices of quacks and charlatans. In addition, the perceived differences in the abilities of doctors trained in different countries are subjected to scrutiny.

At the receiving end of the treatment is the patient. The title of the section, "Good Patients, Better Patients", and the general tone of the excerpts suggest that patients are blameless for their condition; they are seen as good, innocent people wronged by a malady of the mind or body. Nowhere is it found that good patients are those who respond to treatment because the quality of "goodness" is inherent in the endurance of the patients' suffering. One very particular focus in this section is on hypochondriacs, implying that a good deal of patients' illnesses originate in the mind.

It is in the hospitals where most patients are treated, and this is the next area of discussion. This is mostly a discourse on the evolution of hospitals through history and the quality of hospitals in more recent times.

And finally, the concept of the cure is examined, the question being: What matters more, the curing of the patient or the cure itself? Certainly, the wonder of the

cure, the alleviation and cessation of illness is discussed, as well as the effect of the cure on the patient and the ministering physician.

Overall, the book makes very delightful and light reading. Tongue-and-cheek sarcasm nicely punctuates the essential and mostly honourable role to which medicine is ascribed in society. The selection of the anecdotes ranges from hilarious to poignant, yet is always insightful. The book itself is about the size of the **Oxford Handbook of Medicine** but a fraction of the thickness; it can be taken anywhere.

James Kim, 8T9

Manual of Admitting Orders and Therapeutics

Second Edition, W.B. Saunders Company, 1987, 299 pages, $26.75.

Manual of Admitting Orders and Therapeutics is one volume in the "Saunders Blue Books Series" intended to be an aid for primary care physicians, house staff, ward clerks, and medical students. This manual introduces an approach to writing medical orders and was suggested by a former medical student to foster more effective communication in the Doctor's orders. It provides a systematic approach to writing medical orders and this is done by illustrating sample admitting orders for the commonly encountered medical illnesses such as peptic ulcers, MI, diabetes and pneumonia. The orders are accompanied by a narrative that is designed to explain the rationale behind each order. This narrative is placed in a column beside each order for easy access of information. A section on therapeutic agents is included for an up-to-date source of information.

This manual is organized into four parts. Part One discusses the method of writing admission orders and the components of "perfect" admitting orders. Part Two presents examples of admitting orders for over 70 common medical conditions (but no surgical orders). Each order is organized in a convenient format that is easy to follow and read. Part Three contains orders for commonly performed procedures such as arterial line insertion, bronchoscopy, lumbar punctures, etc. Lastly, part Four provides prescribing information (drug dosages, indications, and side effects) for over 500 drugs in the form of a chart. The format throughout the manual is designed for easy and quick retrieval of relevant information and is one of its strong qualities.

The second edition is a completely updated version of the first edition, and major changes include the addition of admission orders for AIDS, toxic shock syndrome, dissecting aortic aneurysms, and other important conditions. Part Three has been increased to include more procedures such as coronary angioplasty. Part Four has also grown to include many new drugs.

Being a third year medical student who is just beginning to learn the skill of writing admission orders, this manual serves as a useful guide in getting acquainted with how they should be written and familiarizes one with items of the orders which may sometimes be forgotten.

Vivienne Ang, 8T9

Clinical Neuroanatomy Made Ridiculously Simple

by Stephen Goldberg, M.D., MedMaster Inc., 1983, 89 pages, 61 illus., $13.95.

There are two new developments that have changed neurology forever. The first: the discovery of the CAT scanner, whereby a patient spending 30 minutes with his head in a 1.5 million dollar machine can have a plethora of diseases either diagnosed or ruled out. The second: Dr. Goldberg's **Clinical Neuroanatomy Made Ridiculously Simple (C.N.M.R.S.),** whereby a medical student can spend less than one day and learn most of the neuroanatomy that is clinically relevant to neurology.

Neuroanatomy is a vast topic that is confusing from the beginning to near end. **C.N.M.R.S.** takes the jungle of neuroanatomy, cuts out all of the heavy and irrelevant brush, and leaves the trees. When studying neuroanatomy for the first time one needs to occasionally refer to a text that provides only the big picture. A prime example of this is the study of the brainstem and spinal cord, a mass of wires arranged by God to confuse students. Frequent reference to illustrations of these pathways will keep this complex area in perspective.

Topics that are not covered extensively are as follows: the internal circuitry of the cerebellum, the thalamus, and the basal ganglia. However, these topics are not often talked about in clinical neurology and are left for the B.Sc. in neuroanatomy (and the first year medical student). A topic that should have been better discussed is the localization of lesions in the cerebral cortex and the syndromes caused by pathology, such as stroke or tumor.

The text is highly recommended to the medical student who is beginning neurology, having left neuroanatomy years ago. A quick review and reference can start one off on the right clinical foot. Using the question-answer section at the end of each chapter and the twenty-four quick cases in the back of the text, the knowledge can be resurrected and reinforced. With a barrage of information being thrown at you from all directions, Dr. Goldberg's sense of humor and unforgettable mnemonics make the information easy to grasp and memorable. An example is his description of the circle of Willis as a spider with a "nose, angry eyebrows, two suckers, eyes that look outwards, a crew cut, antennae, a fuzzy beard, eight legs, a belly that is thin, two feelers on his rear legs, and male genitalia" (where each part represents relevant neuroanatomy of the region).

Dr. Goldberg, in addition to being a neurologist, is an ophthalmologist and a family physician. As a result, he has a vast base of knowledge and an understanding of the fundamentals in many subjects. He has used his teaching skills to write other texts in the same style, a simplistic but relevant one. These other books include: **Clinical Ophthalmology Made Ridiculously Simple, Clinical Anatomy Made Ridiculously Simple** and **The Four-Minute Neurological Exam.** All are enlightening and well-worth having as an adjuvant to the more complete texts.

Sandeep G. Agganval, 8T9

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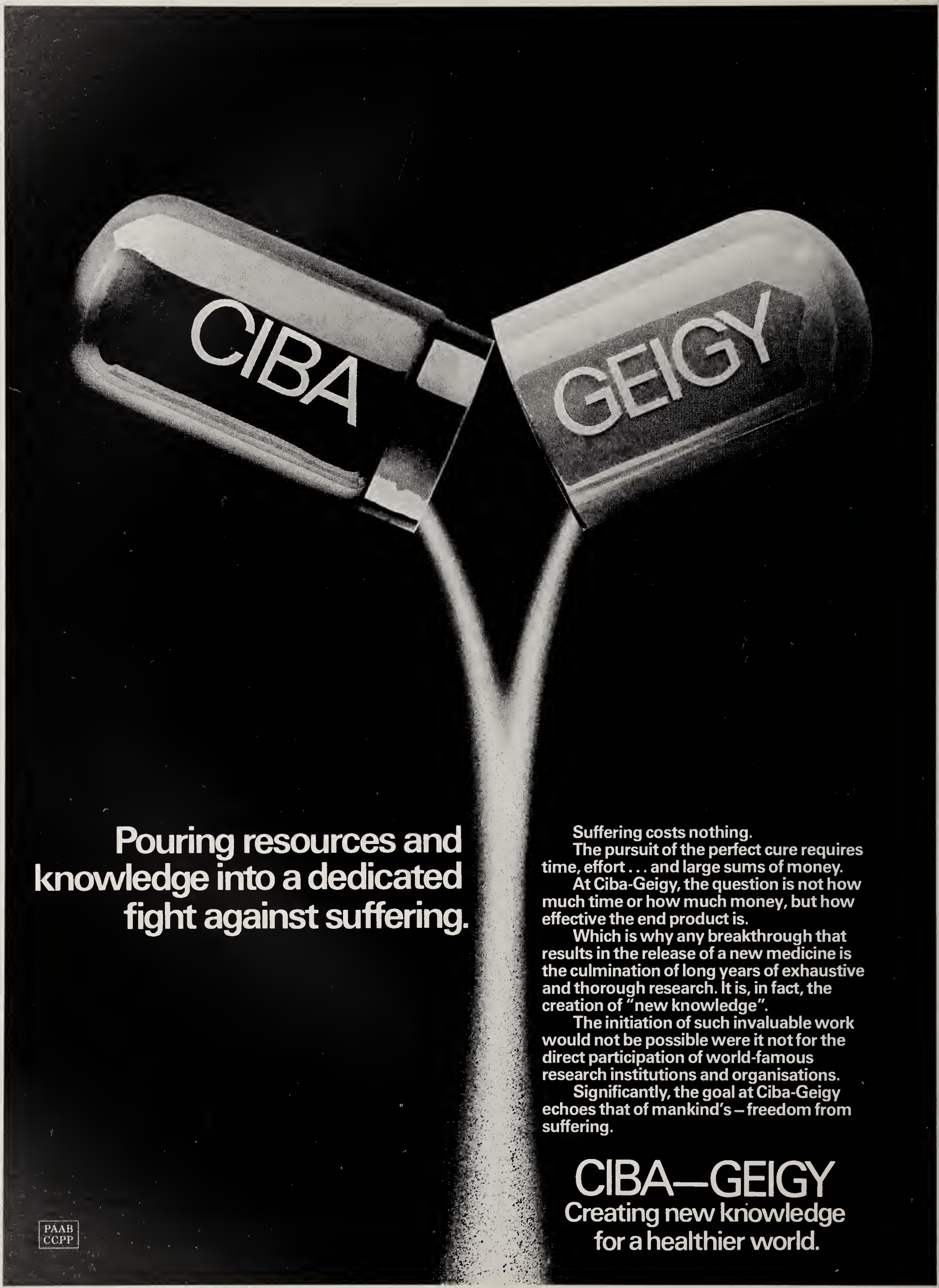

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